VOLUME 50 NUMBER 5

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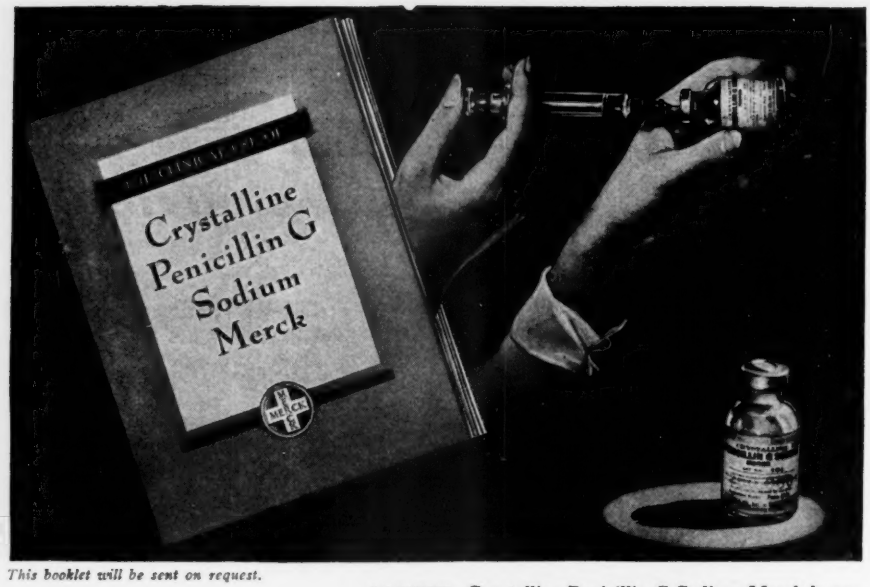

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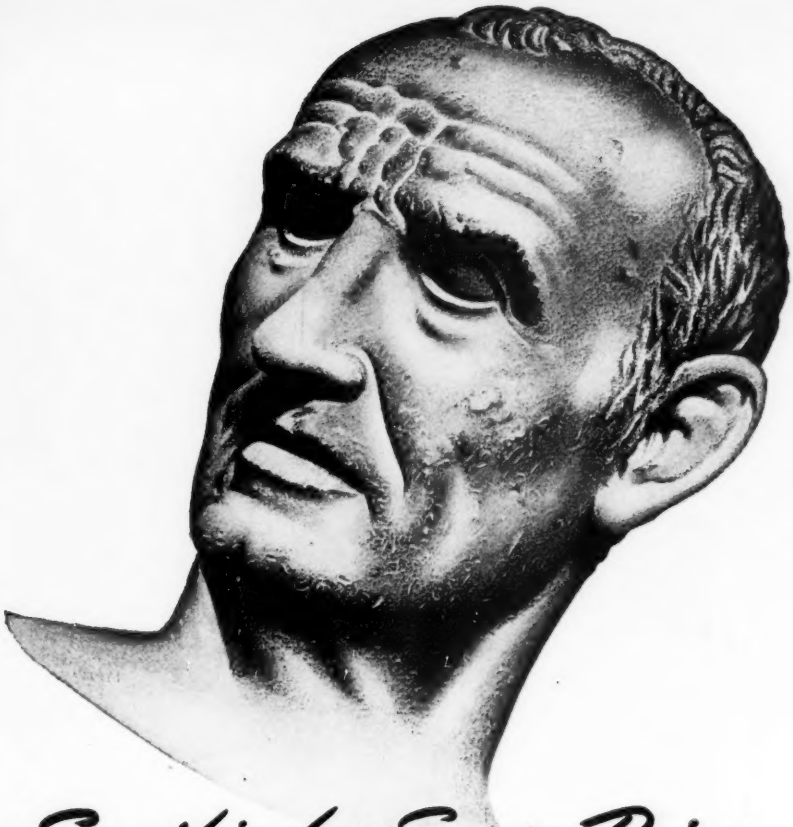

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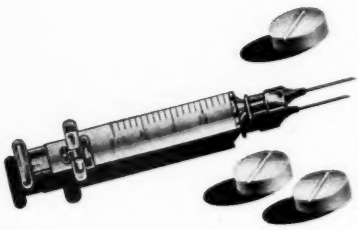

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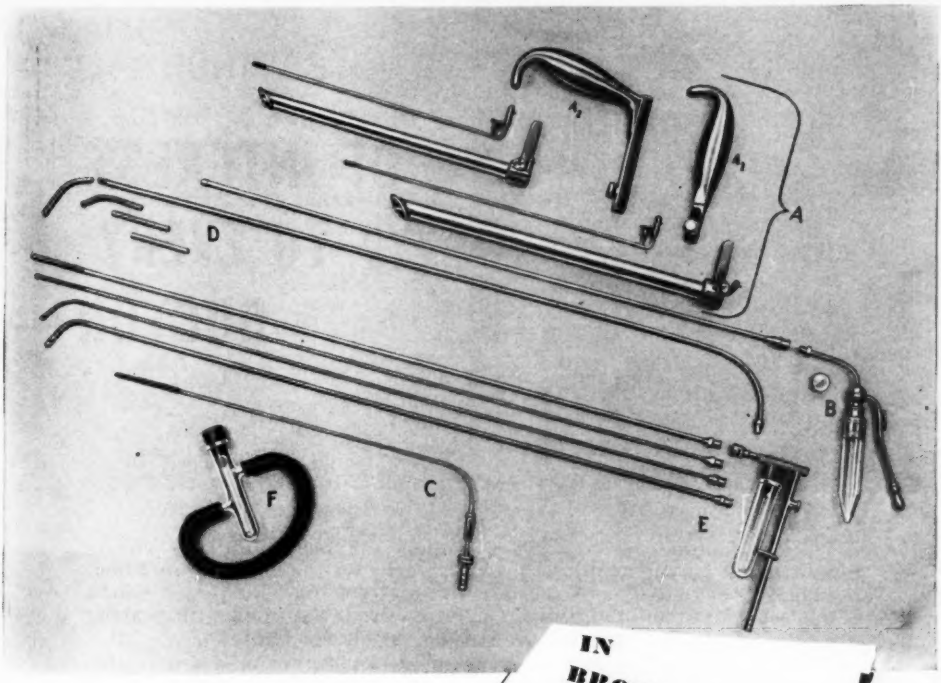

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SURGICAL TREATMENT OF MALIGNANT TUMORS OF THE INFERIOR ALVEOLUS AND MANDIBLE

JOHN J. CONLEY, M.D.
AND
GEORGE T. PACK, M.D.
NEW YORK

THE TREATMENT of choice for primary malignant tumors of the inferior alveolus and mandible is radical resection of the bone and surrounding tissues. The approach and the extent of the surgical procedure are governed by the type, size and position of the tumor. Irradiation is often an invaluable preoperative adjunct.

Tumors of the mandible may be primary or secondary. The primary type arising in the alveolus or mandible comprises the majority of cases. The secondary type may be the result of metastasis from malignant tumors of the prostate gland, breast, kidney, thyroid gland, lung or gastrointestinal tract, or it may be the result of direct extension from a malignant tumor of the lip, tongue, cheek, floor of the mouth or salivary glands. The discussion in this article will be limited solely to primary neoplasms.

Classification of Malignant Tumors of the Lower Jaw

A. Carcinoma
   1. Epidermoid (from gingiva and cheek)
   2. Basal cell (from overlying skin)
   3. Adenocarcinoma (salivary gland tumors)

B. Tumor from bone-forming tissues
   1. Osteogenic sarcoma
   2. Chondrosarcoma
   3. Malignant giant cell

C. Tumor from bone marrow
   1. Ewing's tumor
   2. Myeloma

D. Tumor from neighboring somatic tissues
   1. Fibrosarcoma
   2. Liposarcoma
   3. Malignant melanoma
   4. Lymphosarcoma
   5. Adamantinoma

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E. Tumor from blood vessels and nerves in bone
   1. Angiosarcoma
   2. Angioendothelioma
   3. Neurosarcoma

DIAGNOSIS OF MALIGNANT TUMORS OF THE JAW

The diagnosis of malignant tumors of the jaw is accomplished by (1) clinical examination, (2) stereoscopic roentgen studies of the mandible and (3) biopsy. Malignant growths of the lower jaw are not uncommon. They comprise 20 per cent of all of the cancers of the mouth. They may originate from the soft tissues of the alveolus, the periosteum, the osteogenic substance of the mandible or the nonodontogenic and nonosteogenic substance in the mandible. Although malignant tumors of the alveolus and mandible are situated in readily accessible positions for early diagnosis, most of them are well advanced by the time they are identified and receiving treatment. This is sometimes due to fear or lack of interest on the part of the patient, but very frequently the medical and dental professions are also remiss in making an early and correct diagnosis. Failure to investigate an indefinite type of toothache or pain in the jaw, misinterpretation of inflammatory hyperplasia and ulceration, inattention to precancerous lesions and prolonged observation of hard and soft tumors are some of the reasons for the loss of valuable time in the management of these cases.

In a general way, the carcinomas tend to appear on the alveolus of the horizontal ramus. As a rule they appear after the fourth decade of life. Males are affected more frequently than females. This cancer is more prone to occur in ill kept mouths and as a result of chronic irritation from poorly fitted prostheses. In their early stages carcinomas of the alveolus are usually painless and slow growing, with a tendency toward ulceration and infiltration. After invasion of the mandible, severe pain may be experienced along the distribution of the inferior dental nerve. Occasionally cancer of the jaw may be superficial and fungating in character.

Sarcomas of the mandible may grow very rapidly, have less tendency to ulcerate in the early stages and often cause a dull aching type of pain. They frequently metastasize early to the lungs and other distant organs. All these tumors are firm to palpation except angiosarcoma and lymphosarcoma. Sarcomas may originate inside the mandible, destroy the substance of the bone and perforate into the surrounding soft tissue, or they may originate in the soft tissues and destroy the mandible by invasion. An indefinite aching type of pain in the jaw or teeth is an early symptom. Early and extensive destruction of bone is characteristic. Often the teeth become loose and fall out. There may be a history of trauma preceding the appearance of the tumor in rare

special instances, but a single injury cannot be said to be the causative factor in the origin of these neoplasms.

Stereoscopic examination of the mandible assists in determining the variety and extent of bone destruction. In early malignant tumors of the alveolus, periosteum and bone there is usually no destruction of bone evident on roentgenographic study. In rare instances there may be enough infiltration of bone to cause pain and structural weakness without demonstrable evidence of alteration in density of the bone. However, in the more advanced malignant growths changes in bone are usually obvious. These are manifested by osteolytic and osteogenic alterations in any or all portions of the bone. Invading cancer causes marked reduction in density of the bone without accompanying new bone formation. The infiltration is destructive in character. In contrast, osteogenic sarcoma progresses from an interval position toward the cortex and soft tissues. Roentgenographically there is evidence of osteolytic and osteogenic activity. Fibrosarcoma presents a roughened, eaten-out outline. Chondrosarcoma shows blotchy areas of calcification and ossification associated with a thick shell. Ewing's tumor presents layered, irregular shadows and destruction of bone. Myeloma is characterized by areas of reduced density with essentially normal-appearing adjacent bone. Adamantinoma destroys normal bone and replaces it with multiple cystic bone spaces surrounded with a bony shell. Typical angiosarcoma reveals slight cortical or cancellous erosion and usually a small oval tumor on the periphery of the bone. Sarcoma of the nerves and soft parts about bone may cause pressure erosion.

Biopsy should be done in all cases. In new growths originating on the alveolus, periosteum and external surface of the mandible, biopsy material is readily available. However, it may be necessary in diagnosing a destructive lesion of the medullary cavity of the mandible to open the cortex and secure a direct biopsy at operation. An aspiration biopsy, with use of a 17 gage needle attached to a Record syringe, is often successful in removing sufficient tissue to permit a correct microscopic diagnosis. Usually in all advanced cases of malignant tumors of the mandible there is adequate tissue available for biopsy.

SURGICAL APPROACH

A classification as to the type and extensiveness of involvement of the tissues in the mouth and neck assists in planning the surgical approach.

Class 1: Early, low grade cancer limited to the alveolus

Class 2: Early, low grade cancer limited to the alveolus and upper portion of the mandible

Class 3: Cancer of the jaw which has extended beyond the alveolus or deeply into bone

Class 4: Cancer of the jaw which has extended to the floor of the mouth, cheek and tongue

Class 5: Cancer of the jaw with any degree of local involvement plus metastasis to the upper or deep part of the neck

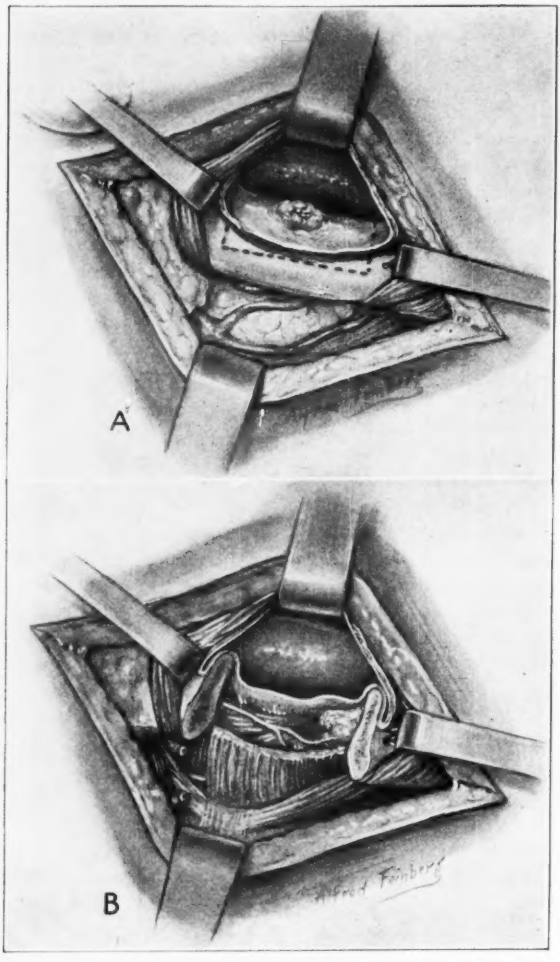

Fig. 1.—*A*, segmental resection of the horizontal ramus for early low grade, limited carcinoma. *B*, cut ends of the mandible, the tongue, the cut border of the mylohyoid muscle and ligated vessels. In this instance the section of mandible has been removed along with the contents of the submaxillary space and tip of parotid gland.

It is imperative in the surgical treatment of neoplasms of the lower jaw that the tumorous growth be widely excised. This resolves itself basically into four types of operations, each with variations and modifications. These technics include the following procedures:

I. Segmental resection of a portion of the ramus with preservation of the inferior rim of the mandible for early, limited cancer of the alveolus. This procedure is not generally associated with the combined mouth and neck dissection (fig. 1*A*).

II. Sectional resection of horizontal ramus with preservation of the ascending rami and usually one horizontal ramus for early cancer of the mandible or alveolus. This procedure may or may not be associated with a suprahyoid or radical lateral neck dissection (fig. 1*B*).

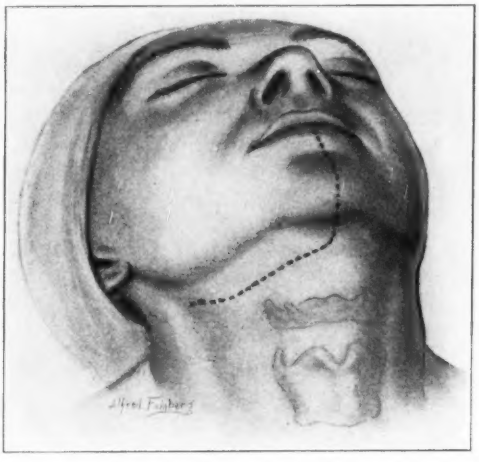

Fig. 2.—Lower lip flap utilized in approach to more extensive cancer.

III. Hemiresection of the mandible with preservation of one horizontal ramus and one ascending ramus for advanced cancer of the mandible with or without extension to the floor of the mouth, tongue and neck. This procedure is usually associated with the combined suprahyoid or radical neck dissection (fig. 2).

IV. Resection of the mandible including both horizontal rami with or without the ascending rami for advanced cancer of the mandible usually with extension to the floor of the mouth, tongue and neck. This procedure is usually associated with the combined resection of the jaw, tongue, floor of mouth and lateral portions of the neck (fig. 3).

All associated soft tissues should be excised with the bone en masse. When the tumor transgresses the boundaries of the jaw, the involved adjacent soft tissues may include a portion of the tongue, floor of the

mouth and buccal mucosa. A further extension of the cancer may appear as metastasis in the suprahyoid or deep lymph nodes of the neck. The attack on cancer of the jaw with metastasis in the neck includes a combined radical resection of the primary cancer which may include tongue, floor of mouth, mandible and cheek, with a radical dissection of the entire neck in continuity. This applies, regardless of the degree of local extension, when any metastatic deposits are discovered in the submaxillary or submental spaces or the deep carotid region.

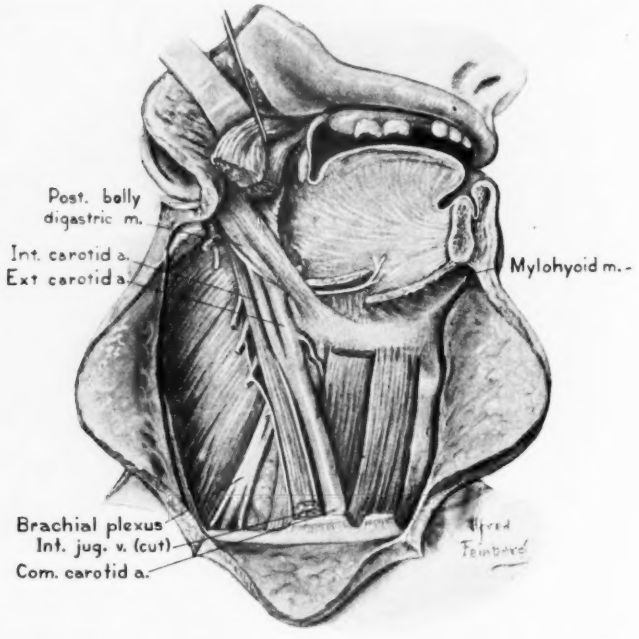

Fig. 3.—Half of the mandible, tongue, floor of the mouth and entire lateral contents of the neck have been removed in continuity. The approach is through the combined incisions of the lateral portion of the neck and lower lip.

In the approach to this surgical problem, certain prophylactic and precautionary measures have been found essential. They reduce the mortality and morbidity and assist in the cure.

Routine ligation of one or both external carotid arteries aids in hemostasis and facilitates the operation. However, it is preferred not to perform an arterial ligation if a bone graft is done in order that the recipient site may retain its blood supply.

Endotracheal or thiopental sodium (pentothal sodium®) anesthesia is preferred.

The pharynx should be snugly packed with gauze packing to prevent the aspiration of blood.

Prophylactic tracheostomy is a safeguard against asphyxiation and should receive serious consideration for all patients in whom the continuity of the mandible is disturbed and the procedure has included manipulation of the floor of the mouth and the neck. When endotracheal anesthesia is employed, the tracheostomy may be done at the conclusion of the operation; when thiopental anesthesia is used, it should be done at the beginning of the operation. Decannulization may be carried out in three or four days.

Installation of an endonasal feeding tube permits maintenance of a normal dietary intake and electrolyte balance.

Oral hygiene should be diligently applied before any radical operation in the mouth is attempted. This is particularly true when grafting is to be done. It includes treatment to infected gums, cleaning and filling of carious teeth and extractions of abscessed teeth.

Energetic use of the antibiotics and chemotherapeutic agents is most helpful in preventing and controlling infection.

Adequate drainage of the wound is necessary.

A large supportive dressing assists in healing.

Intelligent medical supervision of all medical aspects of the case is often the difference between success and failure.

SURGICAL TECHNICS

I. Segmental Resection of the Alveolus and Mandible for Early Limited Carcinoma of the Inferior Alveolus Without Apparent Involvement of Bone and Without Metastasis to the Neck (fig. 1 A).—This neoplasm is treated by a wide intraoral or submandibular excision of the alveolus and underlying bone without fracturing the continuity of the mandible. The buccal and lingual mucous membrane and alveolus are freed 2 cm. or more beyond the gross limits of the tumor on all sides. Gigli saws are threaded over the bone and through two metal directors in soft tissue puncture wounds beneath the mandible when the intraoral route is used. The oral part of the mandible is partially transected proximally and distally to the tumor with the Gigli saw. This segment of bone, periosteum, alveolus and neoplasm is then separated from the lower rim of mandible with a sharp osteotome and mallet. If the segment of bone is anterior, between the mental foramens, it may be removed with the aid of the electric saw. The raw surfaces of the mandible are then covered with appropriate intraoral mucous membrane flaps. Neck dissection is not indicated.

If it has been necessary to leave only a thin rim of the mandible at the site of the excision and there is doubt about its strength and functional ability, intermaxillary wiring should be maintained for six weeks. If the regenerative callus is inadequate, implantation of a bone graft will correct the weakness.

This surgical technic has limited applicability in the surgical treatment of cancer because in the majority of patients presenting themselves for treatment the disease has advanced beyond the stage that intraoral segmental resection will control it.

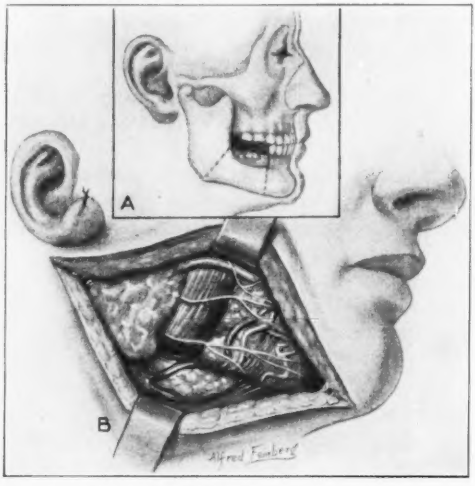

Fig. 4.—Submandibular approach to the horizontal ramus.

II. Sectional Resection for Limited Carcinoma Involving the Mandible or Alveolus Without Metastasis to the Neck, Corrected with Immediate Iliac Bone Graft.—When the alveolus and mandible are involved by an early low grade cancer and there is no evidence of metastasis to the neck, a sectional resection of the bone and alveolus may be performed without neck dissection. This procedure may be followed by an immediate free bone graft in the reconstruction of the mandibular arch. The cancer may be approached intraorally or externally. The external approach may be submandibular or by the creation of a large lower lip flap. When immediate bone grafting is desired, the submandibular approach is preferred. When there is any question of adequate exposure of the tumor, the creation of the lower lip flap is essential.

In the submandibular approach, when bone grafting is desired the soft tissues about the jaw are exposed through a 3 to 4 inch (7.5 to 10 cm.) incision, 1 inch (2.5 cm.) below and parallel to the horizontal ramus. The section of the mandible, with the associated alveolus and periosteum, is freed with the aid of the Gigli saw and double action bone-cutting forceps after appropriate, wide intraoral circumvention of the tumor (fig. 1 *A*, 4 and 5).

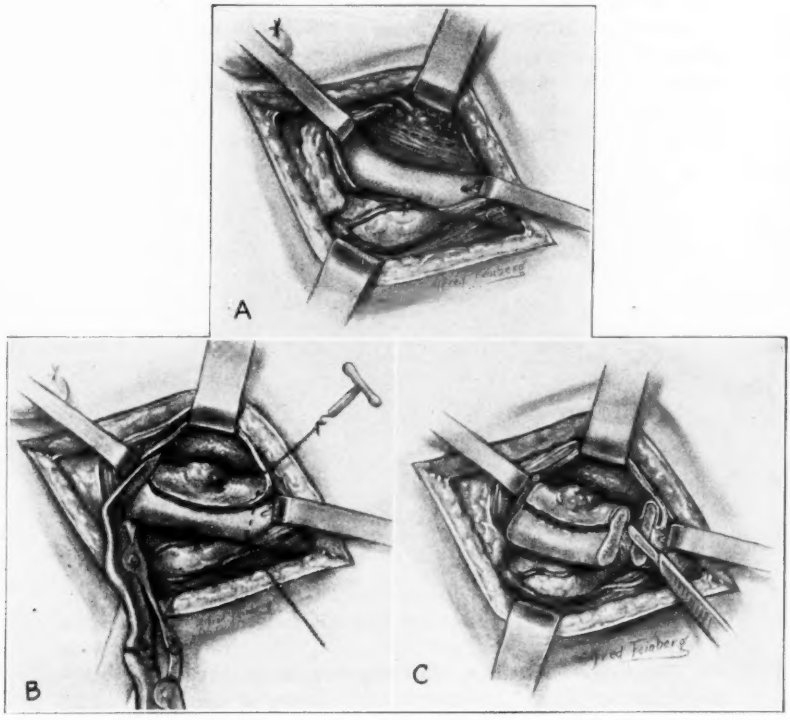

Fig. 5.—*A*, exposure of the horizontal ramus from the mental foramen to the angle with the buccinator muscle still intact. *B*, sectional resection of the mandible. The buccinator muscle is cut. The tongue is visible above and the submaxillary gland below. *C*, freeing the section of the mandible from the floor of the mouth.

An immediate osteoperiosteal graft from the inner aspect of the iliac crest is cut and modeled to duplicate the size and shape of the section of mandible removed.

The methods of fixation of this graft are governed by the number and position of teeth remaining in the mouth and also the position of

the bone graft with respect to the mandibular arch. When a satisfactory number of dental components are available, intermaxillary wiring is the treatment of choice. The major part of the application of these wires may be carried out the day before the operation in anticipation of complete immobility, which is accomplished by the fixation in occlusion of the intermaxillary wires twenty-four hours after the operation. The bone graft in this instance should be wired to the adjoining mandibular surfaces to maintain continuity.

If there are no teeth in the posterior mandibular segment and it is still desired to use the intermaxillary wiring technic, a plaster head cap may be applied and the posterior fragment immobilized by placing a wire through a small drill hole at the angle of the mandible and fixing that wire to a stabilization pole incorporated in the posterior part of

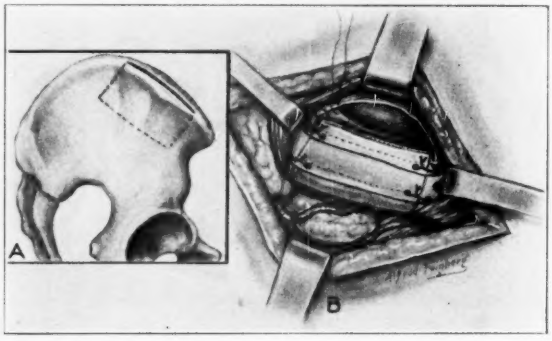

Fig. 6.—*A*, donor site on the iliac crest. *B*, implantation and internal fixation of the free osteoperiosteal bone graft by wiring technic. The submaxillary gland has not been removed.

the head cap. The teeth that are available may be wired together as previously described.

If there are no teeth in either segment, circumferential wiring or the Stador or Roger Anderson apparatus may be used or the graft may be immobilized by internal fixation. The external splints do not work so well practically as might be desired. Frequently the screws become loose, and infection occurs about them. Also, it is difficult to adapt them to the situation in the mandible which has developed as a result of the loss of a large section of the horizontal ramus.

Internal fixation of the graft has proved very satisfactory and may be accomplished by the use of screws, wires or plates. Fixation by wires consists in drilling two holes in each segment of the mandible and at each end of the bone graft. Tantalum or stainless steel wire may then be used to secure each end of the bone graft to the adjacent cut surface

of the mandible, and this may be reenforced by through and through wires supporting the bone graft medially and laterally (fig. 6).

Another technic consists in the use of an internal metal splint. This is modeled from a bone plate to fit the ascending ramus, the free osteoperiosteal bone graft and the cut end of the horizontal ramus. The bone graft is anchored to the free ends of the remaining sections of mandible with braided tantalum wire. The continuity of this arch is then fixed by the application of the modeled metal bone splint, which is held in position with screws in the respective sections of bone (fig. 7).

It is important to cover the bone graft securely above with mucous membrane flaps in the mouth in order to eliminate subsequent contami-

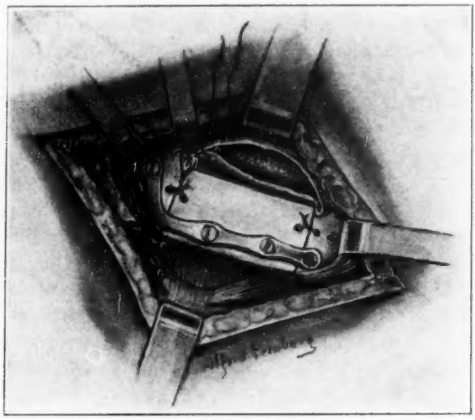

Fig. 7.—Implantation and internal fixation of a free osteoperiosteal iliac bone graft by use of modeled bone plate and screws. The contents of the submaxillary space and the tip of the parotid gland have been excised.

nation and infection. Soft tissues are then adapted about the graft; a small rubber drain is placed in the inferior pole, and a supportive dressing is applied. The patient is fed through a Levin tube for two to four weeks and later given a liquid diet by mouth. The function of the mandible is gradually resumed over a period of another two months.

Variations on this technic include utilization of the rib or tibia or remainder of the mandible as donor sites and fixation with plates, screws and external appliances.

If it has been decided not to do a bone graft at this time, the continuity and position of the jaw may be maintained by internal or external splints or intermaxillary wiring. This mitigates contracture and distortion of the remaining fragments.

A discussion of the treatment of carcinoma of the mandible, at this point, must include the technic of combined suprahyoid or radical neck dissection and excision of the floor of the mouth and part of the tongue. When the carcinoma has extended slightly beyond the alveolus or deeply into the bone, yet without clinical evidence of metastasis in the neck, a suprahyoid dissection should be performed simultaneously with a sectional removal of the bone.

The technic for combined suprahyoid dissection and resection of the jaw may be performed through an extended submaxillary or submaxillary and lower lip type of incision (fig. 2). When the primary carcinoma has extended slightly beyond the alveolus and has definitely involved bone without clinical evidence of metastasis to the neck the approach is limited to a crest-shaped, submaxillary incision extending from the submental area to behind the angle of the jaw. The anterior margin of the mandible is freed with a Gigli saw and the combined specimen of jaw, contents of the submaxillary space and tip of the parotid gland are delivered laterally from the wound. When the carcinoma is in the commonest site, the midportion of the horizontal ramus, this is accomplished by releasing the mandible at the mental foramen and splitting the mylohyoid muscle medially and the buccinator muscle laterally and splitting the deep fascia of the submental space. As the dissection is carried posteriorly, the submaxillary gland and its associated fascia are delivered along with the mandible, its periosteum and mylohyoid attachments. The inferior portion of the parotid gland is included in the specimen. The insertions of the internal pterygoid and masseter muscles are cut, and the specimen is liberated by transecting the mandible again just above its angle. It is usually possible to save the ascending ramus in this type of case, particularly when the primary lesion has not extended to the tongue.

III. Hemiresection of the Mandible for Extensive Carcinoma of the Alveolus and Mandible with Extension to the Floor of the Mouth and Tongue Without Clinical Evidence of Metastasis in the Neck.—When the primary lesion has extended to the floor of the mouth and tongue (and the same applies for a primary lesion of the floor of the mouth or tongue extending to the mandible), yet there is no clinical evidence of extension to the neck, a combined suprahyoid dissection is performed in continuity with the resection of the jaw and tongue. The lower lip is split vertically in its midline, and this incision is connected with the previously described submaxillary incision (fig. 2). The removal of a section of tongue, floor of the mouth and contents of the submaxillary and submental spaces along with the jaw is begun anteriorly by first transecting the mandible with a Gigli or electric saw and continuing the dissection posteriorly as previously described. In cutting the floor of the mouth and tongue it is usually necessary to remove the major

portions of one side of the genioglossus, hyoglossus and mylohyoid muscles and occasionally portions of the anterior belly of the digastric and styloglossus muscles. When the lateral margin of the tongue is involved, the dissection is usually carried posteriorly to the depth of the tonsil. The internal and the external pterygoid, masseter and temporalis muscles and mandibular nerve are stripped from the ascending ramus, and it is avulsed from the glenoid cavity.

Even though it is possible to save the ascending ramus in some of these cases, it is often unwise to attempt immediate reparative and

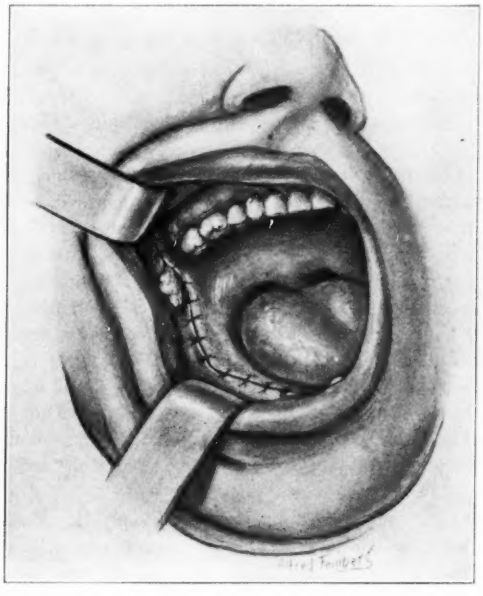

Fig. 8.—Closure of the wound after the intraoral approach for hemiresection of the mandible.

reconstructive bone grafting. This is particularly true when irradiation has been used prior to the surgical procedure. It is well to bear in mind that the prime consideration in all these cases is the cure of the cancer and that all other procedures are of secondary importance. If there are doubts about an immediate bone graft, and there frequently are, the procedure should be eliminated. It may then be done after permanent cure is established. Occlusion of the jaws should be maintained for six weeks with intermaxillary wires to prevent undue distortion of the remaining fragment. Internal appliances are then adapted to pro-

mote a normal bite. In many patients who have not received this special attention, there is often a very satisfactory adjustment to their deformity and eating habits. If the operator has the opportunity and elects to graft, the mandibular joint and a small portion of the ascending ramus should be preserved. This facilitates the grafting procedure and improves the postoperative functional result. A rib or the iliac crest may be used as donor material, or a metal splint may be substituted for a bone graft. Here, as in all grafting procedures, the principles of asepsis and immobilization are imperative.

The intraoral approach may be utilized for the more limited type of cancer requiring hemiresection (fig. 8).

IV. Resection of a Portion or All of the Mandible in Continuity with a Lateral Neck Dissection for Extensive Cancer of the Jaw and Floor of the Mouth with Metastasis to the Neck.—Far advanced cases present any degree of local cancerous involvement plus definite metastatic extension to the upper or deep neck. In all these cases treatment consists in a combined resection of the primary cancer, which may include a portion of the tongue, cheek, floor of the mouth and mandible, in continuity with the complete lateral neck contents en masse. The technic for the combined radical resection of the lateral part of the neck and jaw includes the preparation of four neck flaps containing skin and subcutaneous tissue. This procedure is begun by an incision parallel to the anterior border of the sternocleidomastoid muscle extending from the mastoid bone downward to the clavicle. A modified Z is created by continuing from the midportion of the first incision into the submental area and from slightly below the midportion of the first incision to the lateral border of the clavicle. Undermining the skin beneath these three incisions creates four flaps, which when elevated expose the lateral portion of the neck (fig. 9).

When the carcinoma has not extended to the tongue or floor of the mouth, the mandible may be engaged through the submaxillary and submental approach by the upward elevation of the superior flap. However, when the tongue and floor of the mouth are involved, a vertical incision in the middle of the lower lip is directly connected with the submental extension of the incision in the neck, thus permitting ready access to the entire neck, mandible and mouth. The general specimen is first attacked inferiorly by doubly ligating the inferior part of the external jugular vein and freeing the platysma, sternocleidomastoid and omohyoid muscles at their clavicular levels. After the inferior portion of the internal jugular vein has been doubly ligated, the carotid artery, deep and superficial fascia, internal and external jugular veins, platysma, sternocleidomastoid and omohyoid muscles along with the fat and areolar tissue are rolled upward from the level of the clavicle in their liberation from the deep part of the neck. The thoracic duct should be protected

when the dissection is performed on the left side of the neck. This resection usually permits visualization of the upper portion of the brachial plexus, the scalenus anticus and medius, levator scapula and trapezius muscles. The fifth and sixth cervical nerves are preserved. The phrenic portions of the third, fourth and fifth cervical nerves are maintained intact, but the superficial branches of the second, third and fourth cervical nerves are cut along with the spinal accessory nerve and included in the lateral neck specimen that is being liberated from below upward. At the level of the bifurcation of the common carotid artery, a 4 per cent solution of tetracaine hydrochloride (pontocaine hydrochloride®) is

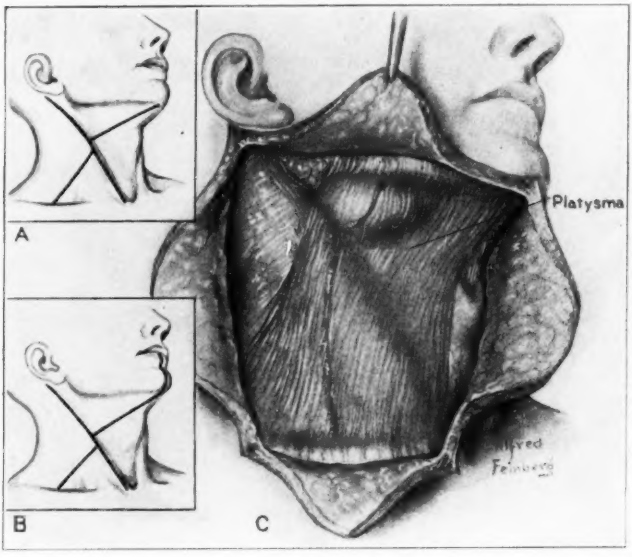

Fig. 9.—*A*, three incisions in the lateral part of the neck for the creation of four neck flaps. *B*, extension of the submaxillary incision to the lower lip for intraoral exposure. *C*, elevation of the neck flaps and exposure of the contents of the lateral portion of the neck.

applied topically and the external carotid artery tied above the superior thyroid branch. It is not necessary to cut the external carotid artery. The mandible is transected well anterior to the primary carcinoma with a Gigli or electric saw. If the primary carcinoma has not extended beyond the mandible internally, the contents of the suprahyoid space and the jaw bone with its periosteum and associated soft tissues are liberated into the wound in the upper part of the neck without cutting the lower lip. A section of the horizontal ramus or half of the mandible may be included in this delivery. The dissection is carried posteriorly,

including the inferior tip of the parotid gland. Care should be exercised to preserve the lingual, hypoglossal and mandibular branch of the facial nerves. Thorough removal of the submaxillary, subdigastric and carotid fasciae is important in this type of cancer. Finally, the origin of the sternocleidomastoid muscle is freed from the tip of the mastoid process, the superior portions of the external jugular, internal jugular and communicating veins are doubly ligated and the combined specimen of jaw and lateral neck content removed en masse (fig. 10).

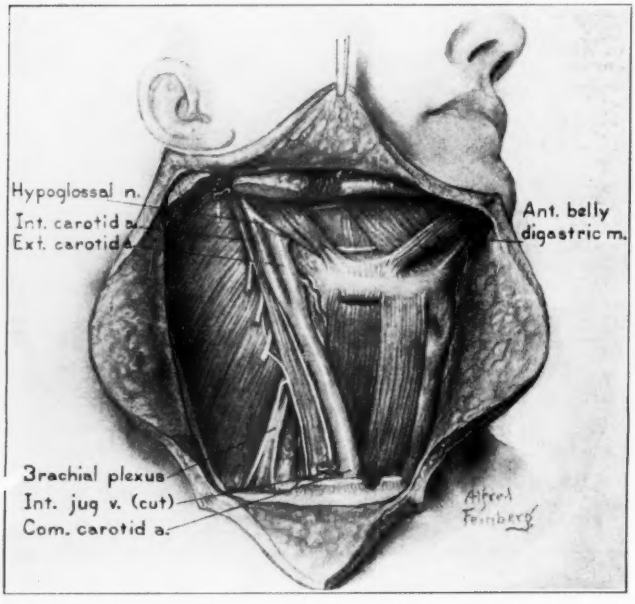

Fig. 10.—The contents of the lateral portion of the neck have been removed.

When the primary carcinoma has extended to the floor of the mouth and tongue, a more open approach and exposure is indicated for the additional resection that is required within the mouth. This is accomplished by connecting a vertical incision in the middle of the lower lip with the submental extension of the incision in the neck (fig. 9 *B*). After the anterior part of the mandible is transected with the Gigli or electric saw, the involved portions of the tongue and floor of the mouth are cut free from the normal oral tissues beginning anteriorly at the tip of the tongue. These additional organs are included in the specimen, along with the bone and contents of the neck as the dissection is carried

posteriorly in the mouth. In the vast majority of these cases disarticulation of the mandible is required, which, as previously described, is accomplished by freeing the ascending ramus of its periosteum, muscle and nerve attachments and forceful avulsion from the socket. The dissection of the tongue and floor of the mouth is as previously described. The point at which the intraoral resection adjoins the mandible posteriorly depends on the size and position of the extension of the carcinoma in the mouth. Usually it is at the depth of the palatoglossus muscle (fig. 3).

Bilateral resection of the horizontal rami and total resection of the mandible are not common procedures. This technic is usually per-

Fig. 11.—Bilateral lower lip flaps are utilized in case of extensive cancer of the jaw and mouth.

formed in cases of extensive malignant tumors of the jaw which have been previously unsuccessfully treated by irradiation and inadequate surgical procedures. Frequently the floor of the mouth, lip and neck have been invaded by the cancer, and it is necessary to remove these organs in continuity with the mandible and neck contents.

A bilateral lower lip flap in association with the neck flaps facilitates the exposure greatly, permitting both horizontal rami to be readily visualized. The lower lip flaps are prepared by splitting the lower lip vertically in the midline to 1 inch (2.5 cm.) below the mandible. The flaps are then freed laterally to the angles of the mandible (fig. 11).

The thickness of these lip flaps is determined by the extent and position of the malignant tumor in the mandible. In bilateral resection of the horizontal rami, the jaw is transected at the angles with the saw or bone-cutting forceps. The freed section is then removed in continuity with all adjacent and involved tissue en masse, as has been described previously. This may include a bilateral suprahyoid or lateral neck dissection. One jugular vein should be preserved, and two to three weeks should elapse between resections of the entire lateral neck contents. We have performed bilateral radical neck dissections with sacrifice of both internal jugular veins within the short interval of ten days.

For complete mandibular resection, one ascending ramus is liberated of all its attachments and avulsed. By manipulating this freed end, the other portion is gradually delivered and avulsed in the manner described. The labial and the buccal mucosa are approximated to the floor of the mouth, and the dead space is eliminated by buried chromic surgical gut sutures. The tongue is shortened. There is considerable recession of the chin, but if the motor function of the tongue and lips is preserved, remarkable rehabilitation may be accomplished in the relearning of articulate speech and in the handling of saliva and food.

The general plan of closure of all wounds of the mouth and neck consists in approximating the cut edge of the buccal mucosa of the cheek with the cut edge of the remaining tissue in the mouth, whether that be the floor of the mouth or the cut border of the tongue. Closure in layers with chromic surgical gut beginning posteriorly and working anteriorly to the midborder of the lower lip is preferred in the elimination of the intraoral fenestra and the reconstruction of a new floor of the mouth. The tip of the tongue should be repaired separately in order to maintain its facility. If a large section of the tongue has been removed with the specimen, the intraoral closure should be reenforced by large through and through sutures. All bleeding is controlled with fine silk ties. The neck flaps are adjusted into their original position and the skin wound is closed with fine interrupted black silk. It is important to maintain adequate drainage at the anterior and lateral inferior portions of the wound. A prophylactic tracheostomy is indicated. A Levin feeding tube is most helpful in maintaining nutrition and also in putting the mouth at rest and facilitating the healing process about the newly constructed floor of the mouth. The remaining mandibular segment is maintained in position by the application of intraoral prostheses after recovery from the operation. Chemotherapy and biotherapy are important in preventing and controlling infection.

Immediate reconstructive surgery is limited in the type of case that has just been described, consisting of radical resection of most of the jaw and contents of the neck. At present, immediate bone grafting is not recommended when the dissection has sacrificed a portion of the tongue, floor of the mouth, jaw and lateral neck contents. It is true

that the most advantageous time to reconstruct the mandible is at the completion of the operation for its removal when this is warranted. An appropriate bed already exists for the implantation of the bone graft. The distortion caused by scar and muscle contracture, which always appears to some degree after the mandible is removed, does not exist. The problem of utilizing a twisted section of the ascending ramus at a later date is averted. The patient is specifically benefited by preservation of the contour of the face and the complete utilization of his mandible as an eating and speaking instrument. The psychic effects of being deprived of these advantages may be serious. Yet, unhappily, it is frequently impossible and unwise to attempt immediate complete reconstruction of the mandible. The factors mitigating the hope of ideal repair are the great loss of bony and soft tissues. Often structures vital to the reconstruction technic must be sacrificed with the specimen. Frequently the size of the resected segment of mandible not only makes the procurement of a similar piece of bone difficult but embarrasses the fixation and immobilization of it. There is always an extensive amount of tissue trauma. The area, because of its association with the oral cavity, is potentially infected. Active infection is often present in the tumor and soft tissues and bone. Often the area has been scarred by inadequate previous surgical procedures. Frequently the tissues have been devitalized by unsuccessful irradiation. However, the strong desire to rehabilitate the patient completely from a functional, psychic and physical point of view is discovering new methods of overcoming the old obstacles. Modeled vitallium and tantalum mandibular inplants may be fixed to the ascending rami when the hazards of a free bone graft are excessive. Perhaps it will be possible to employ prepared mandibles procured from bone banks in the near future in the reconstruction of the more extensive resections. The complete use of the services of the dental prosthetist to assist in maintaining a normal bite and in preventing undue contracture prepares the way for secondary reconstructive procedures.

REPORT OF CASES

No attempt is made in this paper to present a statistical analysis, and a brief summary of only 6 cases representing the various surgical technics described in the article is hereby presented to clarify situations pertaining to each case. Although hemiresection of the mandible is the operation that is most frequently performed, only 1 case of that type will be discussed to illustrate certain features pertaining to the indications and use of that operation.

CASE 1.—*Segmental resection of a portion of the mandible.*

A 57 year old white man originally underwent treatment by the application of radium to an epidermoid carcinoma grade 2 of the lip in 1931, 1934 and 1937. In

1944 the lip and chin were again treated with the application of radium and also by roentgen therapy. The patient was then seen in this clinic, and examination revealed a large, ulcerating and perforating defect in the lower lip and chin, solidly infiltrated with carcinoma. The gingivolabial gutter was involved by this carcinomatous process, and the lesion was firmly adherent to the periosteum at this site. The lower incisor teeth were necrotic and loose. Stereoscopic examination of the jaw revealed apical abscesses of these teeth but no specific evidence of cancer.

The surgical approach to this problem included the entire resection of the remainder of the lower lip and chin along with the periosteum and upper portion of the mandible at this site. It was possible in this case to free the periosteum from the inferior border of the mandible between the mental foramens superiorly, approximately ½ inch (1 cm.). The upper portion of the mandible containing the teeth was freed with a horizontal incision and two vertical incisions in the bone. The bone, teeth, periosteum and soft tissue about the chin and lip were

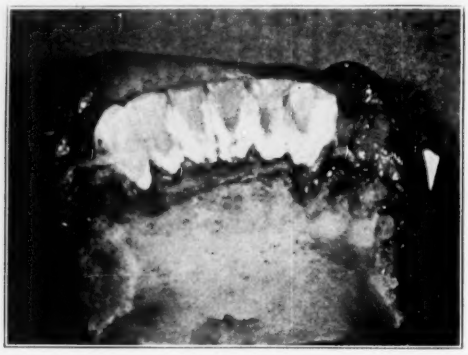

Fig. 12.—Segment of the anterior portion of the mandible associated with the lower lip. The inferior rim of the mandible has been preserved.

removed en masse. A thin rim of mandible between the mental foramens, measuring slightly less than ½ inch in width, was preserved inferiorly. This bone appeared perfectly healthy. There was no evidence of metastasis to the neck. A new lip was immediately created by transposing lateral cheek and mucous membrane flaps medially over the preserved arch of the mandible. The postoperative course was uneventful, and the patient has been free of disease for two and a half years (fig. 12).

CASE 2.—*Sectional resection of the mandible with immediate iliac bone graft.*

A 58 year old white man presented a grade 2 squamous cell carcinoma of the posterior portion of the right alveolus. The patient's history revealed that the third molar tooth became loose in the socket and was extracted eight months before he visited this clinic. This socket failed to heal, and a small ulcer appeared. Examination revealed a small depression over the socket of the third molar on the right side of the mandible manifesting a tendency to heal over completely, which is unusual in the mouth. It measured approximately 1.75 by 0.75 cm. and was directly

over the socket. Both gutters were free. There was no evidence of metastasis to the neck. Stereoscopic examination of the mandible failed to reveal a cancerous process in the bone.

Surgical procedure included the complete release of a section of the mandible at this site, measuring approximately 6 cm. in length and including the bucco-

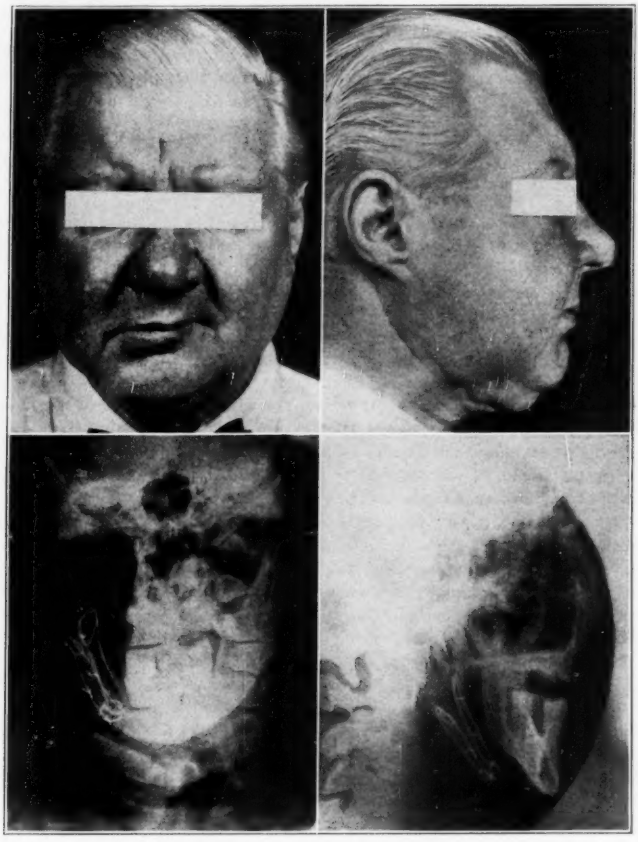

Fig. 13.—View of the face and free bone graft held in position by tantalum wire one year after operation.

alveolar gutter and the glossoalveolar gutter and their soft tissue and mucous membrane contents. The surgical approach was through a crescent-shaped incision in the right submaxillary space. The submaxillary gland was not resected. Immediate repair and reconstruction were accomplished by the use of a free osteo-

periosteal iliac bone graft which was modeled to correspond to the size and shape of the section of mandible that was removed. It was fixed in position by two tantalum wires through the ascending ramus and the posterior segment of the bone graft and two tantalum wires through the anterior section of the bone graft and the anterior free edge of the mandible. It was further immobilized by passing two tantalum wires completely around the internal and external surfaces of the graft and fixing these wires to the remaining sections of mandible. No external fixation was utilized. The patient was fed with a Levin tube for approximately a month and was then given a liquid diet for the second month and gradually rehabilitated over the next six weeks. The postoperative course was uneventful, and the patient has been free of disease for one and a half years (fig. 13).

A second case of carcinoma of the right alveolus and horizontal ramus is presented because of the utilization of a different technic in the sectional bone grafting procedure.

CASE 3.—*Sectional resection of the horizontal ramus in continuity with the submaxillary space contents and immediate reconstruction with free iliac bone graft and internal fixation.*

A 48 year old white woman revealed that a whitish, thick nonulcerative area had existed underneath a poorly fitting dental prosthesis for several years. She was aware that this had been ulcerated for approximately six months. Examination revealed a poorly fitting prosthesis on the mandible, moderately advanced leukoplakia of the right alveolus and a grade 2 epidermoid carcinoma of the midportion of the right alveolus and ramus. It measured approximately 2 cm. in length and 1.5 cm. in width. The buccoalveolar and glossoalveolar gutters were free. There were no abnormal masses in the neck. The lesion had deeply ulcerated into the bone. Stereoscopic studies of the mandible revealed erosion of a section of the superior portion of the horizontal ramus on the right side at the site of the carcinoma.

The mandible was approached through a crescent-shaped right submaxillary skin incision, and a right cheek flap was created external to the platysma muscle well above the buccoalveolar gutter. A portion of the sublingual gland and the contents of the submaxillary space were incorporated in the liberation of the internal portion of the mandible. This section of mandible, periosteum, alveolus, mucous membrane of buccoalveolar and glossoalveolar gutters, platysma, a portion of the musculature of the floor of the mouth and a section of the sublingual gland and contents of the submaxillary space were removed en masse well beyond the gross limits of the carcinoma. Immediate reconstructive procedures were performed with utilization of a free osteoperiosteal iliac bone graft which was modeled to correspond to the size and shape of the section of mandible removed. The intraoral fenestra was closed first in double layers with no. 1 atraumatic intestinal chromic surgical gut. The bone graft was then affixed to the ascending ramus and the anterior section of the mandible with braided tantalum wire through holes in the graft and the sections of mandible. Complete immobility and fixation of the archway was then accomplished by the application of a modeled stainless steel bone plate which was previously prepared to fit this reconstruction. With the aid of the bone plate the graft was securely fixed in position with two screws and then anchored to the ascending ramus with one screw and to the anterior segment of the mandible with another screw, thus fixing the two free segments of the mandible, the bone graft and the bone plate into a permanent, completely stabilized

mandibular arch. This patient was operated on six months before the time of this report. Examination of the surgical specimen did not reveal cancer in any of the cervical lymph nodes.

This technic of reconstruction is, in general, superior to the other technic because of the absolute fixation of the bone graft and the more rapid rehabilitation of the patient (fig. 14).

Fig. 14.—View of face and free bone graft held in position with a modeled stainless steel plate and screws three months after operation.

CASE 4.—*Hemiresection of the mandible and suprahyoid dissection in continuity.*

A 52 year old white woman stated that she had been aware of the presence of a tumorous mass on the right side of her jaw for approximately eighteen months before she sought medical aid. Examination revealed a chronically infected, deeply ulcerating, foul-smelling, epidermoid carcinoma grade 2, measuring approximately 4 cm. in length and 2 cm. in width in the right glossoalveolar sulcus involv-

ing the alveolus, the internal part of the mandible and the adjacent floor of the mouth. It was associated with the horizontal ramus. There were no teeth at this site. There was no evidence of metastasis to cervical nodes. The surgical procedure was performed through a submaxillary incision and included ligation of the right external carotid artery and hemiresection of the right side of the mandible including the floor of the mouth, a small section of the tongue, the palatoglossus fold, the submaxillary gland and a portion of the sublingual gland and tip of the parotid gland. The intraoral fenestra was closed with no. 1 chromic surgical gut in layers, the margin of the tongue being sutured to the cut edge of the buccal mucosa. The wound in the neck was closed with fine black silk, with a drain in the inferior pole. There was no evidence of cancer in any of the lymph nodes of the neck. The postoperative course was uneventful, and there was no evidence of cancer twenty-eight months postoperatively.

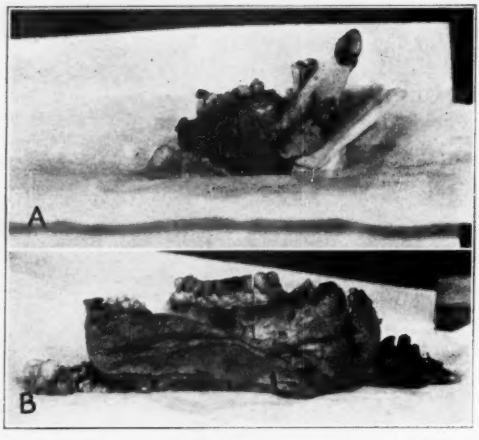

Fig. 15.—*A*, specimen containing half of the mandible, tip of the parotid gland, contents of the submaxillary space, sublingual gland, floor of the mouth and lateral margin of the tongue. *B*, resection of both horizontal rami and left ascending ramus with bilateral radical suprahyoid dissection and resection of associated skin. (This specimen had been fixed in formaldehyde solution U. S. P. and sections removed for analysis before this photograph was taken.)

This case illustrates the treatment of a more advanced type of carcinoma, including the radical resection of not only the bone but also a portion of the associated soft tissues. The continuity of the mandibular arch is maintained by intermaxillary wiring and the application of appropriate intraoral bars and splints to the maxilla and remaining section of the mandible. Complete rehabilitation and orientation of persons who have undergone this type of operation are considerably more difficult than of patients receiving immediate reconstructive procedures, including bone grafting (fig. 15 *A*).

CASE 5.—*Bilateral resection of the horizontal rami and the ascending ramus on one side with bilateral suprahyoid dissection in continuity.*

A 58 year old white man revealed that he had been treated for carcinoma of the lower lip six years before the time of this report. Approximately two and a half years ago he became aware of a swelling in the left submental space. After a formal biopsy this was treated by roentgen irradiation and the application of radium. After the interval of a year tumors began to appear on both sides of the site of the original mass in the neck. These were removed by limited surgical intervention. Shortly before visiting this clinic, the patient noticed that the tissues about the jaw were beginning to swell again. Examination revealed a post-irradiation scar on the left lower lip but no evidence of recurrence of the epithelioma at this site. The tissues about both horizontal rami were diffusely involved with metastatic deposits of cancer. The upper part of the neck and the floor of the mouth were markedly indurated as a result of the previous surgical procedure and irradiation. The infiltrative process about the upper part of the neck had invaded the periosteum and bone of both horizontal rami. Stereoscopic examination of the mandible failed to reveal the invasion of the bone by the carcinomatous process. However, microscopic report on the tissues of the upper part of the neck and mandible revealed massive direct extension of squamous cell carcinoma grade 2 into all of the soft tissues about the mandible and also into the mandible. The surgical technic in this case was accomplished by a crescent-shaped bilateral suprahyoid incision with the creation of bilateral cheek and chin flaps. The entire mandible, except a portion of the right ascending ramus, was removed in continuity with a bilateral suprahyoid dissection. The muscular activity of the lower lip and also the tongue were maintained intact. The wound was closed by suturing the under surface of the tongue to the lateral cheek wall with interrupted no. 1 chromic surgical gut in layers, forming a new floor of the mouth. A prophylactic tracheostomy was performed. The postoperative condition was characterized by the sincere efforts of this patient to relearn a new method of handling food and saliva and also learning to speak intelligibly. If the motor function of the lips and tongue is maintained, this is accomplished without too much difficulty. Complete reconstruction in this type of case should be delayed because of irradiation effects on the tissue, postsurgical scar formation, the extensive sacrifice of tissue which is necessary to eradicate the carcinoma and the extensiveness of the carcinomatous involvement. This patient is well two years following his operation (fig. 15 *B*).

CASE 6.—*Hemiresection of the mandible and floor of the mouth and partial glossectomy in continuity with complete lateral neck dissection.*

A 55 year old white man stated that a painless ulcer had appeared on the right side of his jaw bone approximately seven months ago. It spread to the floor of the mouth and was then treated by roentgen therapy. The major portion of the ulcer disappeared, but recently the jaw became painful and a lump appeared in the neck. Examination revealed a large squamous cell grade 3, ulcerated cancer of the right horizontal ramus with extension to the floor of the mouth and one lymph node containing metastatic cancer in the submaxillary space. Stereoscopic roentgenograms revealed destruction of bone of the horizontal ramus.

The surgical approach consisted in the creation of the lateral neck and lower lip flaps. This was accomplished by connecting the vertical incision in the middle of the lower lip with the submental extension of the neck incisions.

A complete right lateral neck dissection was performed up to the hyoid bone. Then the mandible was transected anteriorly and liberated in conjunction with the floor of the mouth, a section of tongue, sublingual gland and contents of the submaxillary space contents. The tip of the parotid gland was cut off and the subdigastric space carefully dissected. The superior portion of the internal jugular vein was ligated and the sternocleidomastoid muscle freed from the tip of the mastoid process. All muscular and nerve attachments of the ascending ramus were stripped and the mandible avulsed from the socket. A combined specimen of lateral neck contents, portion of the tongue, floor of the mouth and mandible was removed in continuity. The wound was closed as described in the text. The postoperative course was uneventful and the patient has been free of disease for two years (fig. 16).

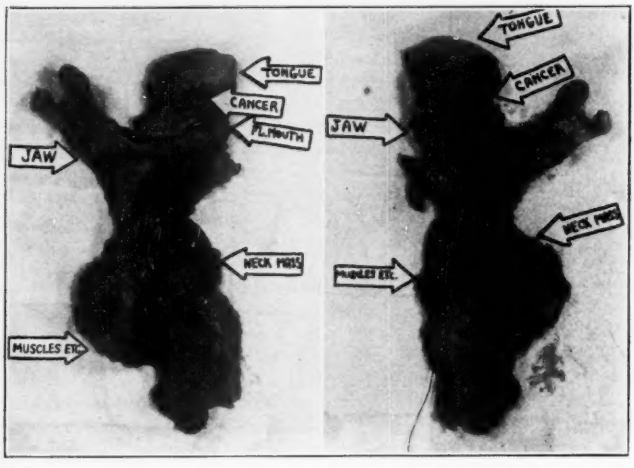

Fig. 16.—Specimen containing a portion of the tongue, floor of the mouth, half of the mandible and entire lateral neck contents.

SUMMARY

1. A classification of the primary types of malignant tumors of the mandible is presented.

2. A classification of the various types of cancer of the mandible in respect to size, position and extension is stated.

3. Four basic surgical technics with individual modifications are recommended for the treatment of these cases.

4. Immediate reconstructive surgery is discussed and qualified.

5. Six illustrative cases are reported to clarify the analysis and surgical approach to the various degrees and types of cancer of the mandible.

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SURGICAL CORRECTION OF THE ABNORMALLY PROTRUDING EAR

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NUMEROUS methods of correcting a protruding auricle have been described, and in the main they are satisfactory for setting the ear closer to the head. However, the object of restorative surgery for any deformity is to reproduce the normal as accurately as possible, a standard which most accepted technics of otoplasty fail to fulfil.

An examination of some of the more recently published procedures for the correction of outstanding ears reveals a gradual progression toward the goal of attaining perfection of results. The purpose of this paper is to present an original method of otoplasty which in my experience has yielded results that more satisfactorily simulate the normal, to discuss the underlying factors in the etiology and development of protruding ears and to review the history of the operative procedures.

ETIOLOGIC FACTORS

Malformations of the auricle are not unusual and may range in severity from complete absence to macrotia. By far the commonest deformity is the protruding ear.

The embryonic origin of the external ear is still a controversial matter, but most observers agree that the six tubercles, or hillocks, of the first branchial groove, which appear about the fifth week of embryonic life, develop into the pinna. The history of these hillocks and their role in the formation of the auricle have been evaluated by a series of investigators, namely, His, Gradenigo, Schwalbe,[1] Baum and Dobers, Henneberg, Streeter [2] and others. The consensus is that the hillocks are gradually fused and transformed into the adult ear. For convenience, these hillocks are numbered from 1 to 6; three

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1. Schwalbe, G.: Die Ohrmuschel, in Handbuch der Anatomie des Menschen, Jena, G. Fischer, 1898, vol. 5, p. 113.

2. Streeter, G. L.: Development of the Auricle in the Human Embryo, Contrib. Embryol. **14**:111-138, 1922.

hillocks develop on the mandibular arch and three on the hyoid arch of the first branchial groove. Below the hyoid arch a fold of the hyoid integument is formed and is known as the auricular fold. A similar fold appears below the first branchial groove and unites with the auricular fold to form the free margin of the auricle. According to Schwalbe,[1] the points of fusion of these two folds mark the position of the satyr tubercle. Darwin's tubercle occurs about the middle of the margin of the free auricular fold and corresponds to the apex of the auricle in lower mammals. The tragus is derived from mandibular hillock 2; the helix, from mandibular hillocks 2 and 3; the anthelix, from hyoid hillocks 4 and 5, and the antitragus, from hyoid hillock 6. The lobule represents the lower end of the auricular fold.[3] Hammar[4] stated the opinion that the scaphoid fossa is the result of the closure of the branchial cleft.

Wood-Jones and Chuan[5] stated the belief that the pinna is a completely hyoid derivative and that the mandibular contribution is represented only by the tragus and the anterior part of the external auditory meatus. They based their conclusions on the finding of an absence of the tragus in cases of micrognathia or complete agnathia in the presence of a well formed pinna. Further evidence to substantiate their conclusions is based on my own observations in cases of congenital absence of the ear. In all these cases of absence of the auricle in the presence of a well formed mandible, evidence of a tragus and lobule was found.

At the end of the third month of embryonic life the hillocks are well defined and the ear begins to assume definite form. According to Evans,[6] it is at this time that the greatest number of malformations occur. Fraser,[7] however, stated the opinion that it is during the second month of embryonic life that maldevelopments appear. The ear margin at this time is pointed, and, because the crura of the anthelix are not formed, the ear protrudes from the head. Davis and Kitlowski[8]

3. Streeter.[2] Arey, L. B.: Developmental Anatomy, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

4. Hammar, J. A., cited by Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose and Throat, Baltimore, Williams & Wilkins Company, 1947, p. 62.

5. Wood-Jones, F., and Chuan, W. I.: Development of the External Ear, J. Anat. **68**:525-533, 1934.

6. Evans, H. M.: The Origins of Hearing—Random Variation or Convergent Evolution—Study of Auditory Organ and Its Swim-Bladder Connections in Fishes, J. Laryng. & Otol. **50**:649-670, 1935.

7. Fraser, J. S.: Maldevelopment of the Auricle, External Acoustic Meatus and Middle Ear: Microtia and Congenital Meatal Atresia, Arch. Otolaryng. **13**:1-27 (Jan.) 1931.

8. Davis, J. S., and Kitlowski, E. A.: Abnormal Prominence of the Ears: A Method of Readjustment, Surgery **2**:835-848, 1937.

stated that about the sixth fetal month the margin curls, forming the helix, the anthelix becomes more definitely folded and its crura appear. It is the folding of the anthelix and the development of the crura that are responsible for bringing the ear closer to the head.

The protruding ear, therefore, is embryonically a congenital deformity in which hyoid hillocks 4 and 5 are involved. It is inherited according to mendelian law and may be dominant or recessive. Heredity is a factor which occurs repeatedly in many of my series of cases and in cases reported in the literature. Potter [9] reported that a dominant gene may be transmitted through many generations. She revealed the transmission through five generations of a malformation in which the ears were extremely cupped and protruding. A family tree embracing 92 members was obtained in the study. On the average, half of the members with this deformity had transmitted it to their children, while normal siblings married to normal persons had not transmitted the defect.

The cause of the malformation is a hereditary one, but in rare cases intrauterine injury may account for a mild deformity of the auricle. An umbilical cord twisted around a child's head may be the responsible factor in producing a unilateral deformity of the ear.[7]

Imhofer [10] stated the opinion that certain peculiarities of formation of the auricle are an important aid in the establishment of descendants, as, for example, in cases in which the parentage of a child is in doubt. In a few of my cases a mother and child had the identical type of protruding ears. Tracing the family pedigree in one of these cases revealed that the mother was one of four children, of whom a brother had a similar deformity, while the other two siblings, a brother and a sister, were free of any deformity. The father in this case had no auricular deformity, but the mother had protruding ears. In the majority of my cases a history of a similar deformity in some other member of the family was obtained.

CLINICAL SIGNIFICANCE

Since little importance can be ascribed to the function of the auricle, it is only because of the deleterious psychic effect on the patient that protruding ears assume a clinical significance. Actually, according to comparative anatomists, the external ear in man is undergoing regression. In comparing the auricular muscles in man with those of animals with ears of similar type, a definite decrease in size, even to atrophy, is evident. In lower animals the auricle has real function in determining

9. Potter, E. L.: A Hereditary Ear Malformation Transmitted Through Five Generations, J. Hered. **28**:255-258, 1937.

10. Cited by Politzer, A.: A Text-Book of the Diseases of the Ear, London, Bailliére, Tindall & Cox, 1909.

direction of sound and in protecting the internal auditory mechanism. Some animals, on stimulation of the tragus, will close the auditory canal by an infolding of the auricle to serve as a protection against water or insects. Henneberg [11] and others observed that some mammals of the aquatic and semiaquatic group, such as the muskrat, seal and beaver, can close the external ear when they are irritated by mechanical or electric stimulation or when plunged into water. Henneberg observed that the Norwegian brown rat, which is a semiaquatic mammal, after coming out of the water shakes its head violently before opening its ears. If prevented from shaking its head by being held at the nape of the neck, it refuses to open its ears. The closure is affected by pulling the tragus and antitragus together, as well as the anthelix and scapha, so that the external meatus is effectively closed. Rodents, moles and other ground-burrowing animals are also capable of closing the external meatus.

Of all the depressions on the anterior surface of the auricle, it is principally the concha which collects a portion of the sound waves and reflects them into the meatus. Schneider [10] proved that a slight decrease in hearing power takes place if this depression is filled with wax. The size of the auricle and the angle formed with the head also have a slight influence on the reflection of sound into the meatus. This is evident from the fact that persons of normal hearing, as well as those who are hard of hearing, hear somewhat better if the auricle is bent forward or is augmented by cupping the hollow of the hand over it. Although no real difficulty in hearing results from loss of the auricle, nevertheless, sound will not be perceived as distinctly and fully as though the auricle were present.

The psychic effects of protruding ears vary with the sensitivity of the individual person. In almost all my patients, and, surprisingly enough, the effect was pronounced in children, a feeling of insecurity was evident, since this deformity, unlike most malformations, produces reactions of mirth in other people. To be constantly reminded of and ridiculed about a deformity will break all defense mechanisms of repression which the patient might have formed and subjects him to the development of an insecurity or inadequacy neurosis. It is fortunate for these persons that this deformity can be corrected simply and satisfactorily, preferably at an early preschool age, before any real psychic trauma can be inflicted. Many who appear outwardly capable of rising above some defects may be sensitive about them and suffer inwardly. Other patients who are not at all sensitive about their appearance or defects may desire a correction of the ears purely for economic or social reasons.

11. Henneberg, B.: Ueber die Bedeutung der Ohrmuschel, Anat. Hefte **40**:95-147, 1909-1910.

ANATOMIC AND PATHOLOGIC FEATURES

The normal ear is a thin, shell-like structure, having definite depressions and projections. Many variations are found which may still be considered within normal limits. It is only when the ear protrudes abnormally or is absent that the defect is immediately noticeable. Very large or very small ears which may be proportionately more deformed than a protruding ear may go unnoticed if they are placed in a normal relation to the head.

The normal auricle, or pinna, is described by Gray [12] as ovoid with its larger end directed upward (fig. 1). The prominent rim of the auricle is called the helix. Where the helix turns downward, a small projection, known as Darwin's tubercle, is frequently seen. This tubercle is very evident about the sixth month of fetal life, when the

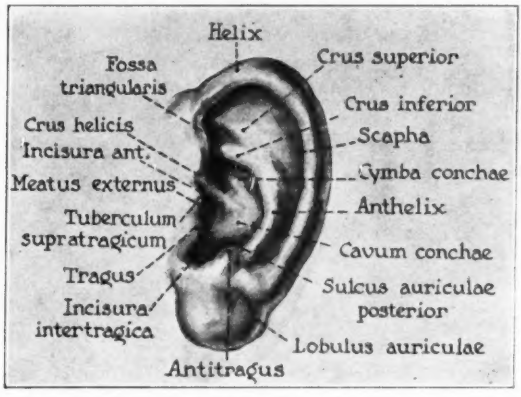

Fig. 1.—The normal auricle and landmarks. The rounded contour of the crus superior is noted, along with the sharper outline of the anthelix and inferior crus. In the protruding ear, the anthelix and its crura are poorly formed, and often the superior crus is absent. This factor, plus a prominent cauda helicis in many cases, accounts for the abnormally protruding ear.

whole auricle has a slight resemblance to that of a macaque monkey. This fact has caused much speculation and controversy among anthropologists, but as yet the significance of this tubercle is not clear. Another prominence, parallel with and anterior to the helix, is called the anthelix; this divides above into two crura, the superior and the inferior, between which is a shallow triangular depression, the fossa triangularis. Some authors refer to these two crura as the posterior and the anterior crus. The narrow curved depression between the

12. Gray, H.: Anatomy of the Human Body, edited by W. H. Lewis, ed. 24, Philadelphia, Lea & Febiger, 1942.

helix and the anthelix is called the scaphoid fossa, or "boat-shaped ditch." The anthelix describes a curve around a deep cavity, the concha, which is divided into two parts by the crus, or commencement of the helix; the upper part is termed the cymba conchae, the lower part the cavum conchae. Anterior to the concha and projecting back over the external auditory meatus is a small pointed eminence, the tragus, so called from its being generally covered on its under surface by a tuft of hair, resembling a goat's beard. Opposite the tragus, and separated from it by the intertragic notch, is a small tubercle, the antitragus. Below this is the lobule, which is composed of areolar and adipose tissue. At the junction of the anthelix and the antitragus a small space exists, which may be very deep in some ears and is known as the sulcus auriculae posterior (Spalteholz [13]).

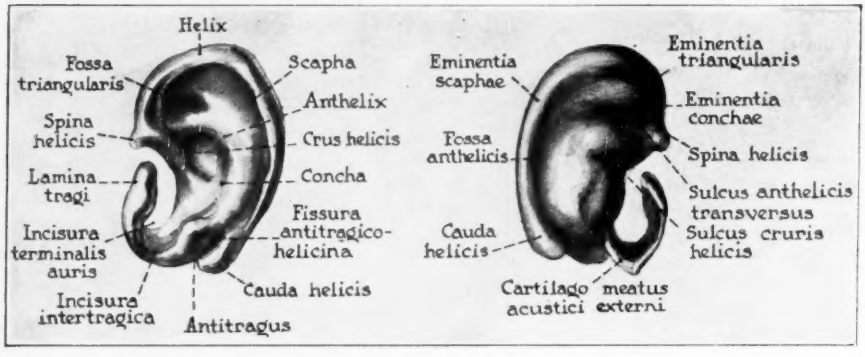

Fig. 2.—The normal auricular cartilage and landmarks from the anterior and the posterior surface.

The auricle, with the exception of the lobe, is composed of a thin, flexible plate of yellow elastic fibrocartilage. The cartilage is 0.5 to 1 mm. thick and is covered on each surface by integument intimately applied and having a minimum of subcutaneous tissue. The skin is closely adherent to the surface of the cartilage except on its posterior surface and along the helix.

The cartilage of the auricle consists of a single piece, which gives form to the ear and outlines the landmarks previously mentioned (fig. 2). It is absent from the lobule and is deficient between the tragus and the beginning of the helix. At the anterior part of the auricle, where the helix bends upward, is a small projection of cartilage, called the spina helicis. At the lower part of the helix the cartilage is

13. Spalteholz, W.: Hand Atlas of Human Anatomy, Philadelphia, J. B. Lippincott Company, 1923.

prolonged downward as a tail-like process, the cauda helicis, which is separated from the anthelix by a fissure, the fissura antitragohelicina. The cranial aspect of the cartilage exhibits a transverse furrow, the sulcus anthelicis transversus, which corresponds to the inferior (anterior) crus of the anthelix and separates the eminentia conchae from the eminentia triangularis. Another furrow exists at the central region of the concha, known as the sulcus cruris helicis, which corresponds to the continuation of the helical rim and separates the concha into two portions, the cavum conchae and the cymba conchae.[13]

The ligaments of the auricle consist of two sets: (1) extrinsic, which connect the auricle with the side of the head, and (2) intrinsic, which connect various parts of its cartilage together and to the external auditory meatus.

The muscles of the auricle likewise consist of two sets: (1) extrinsic, which connect it with the skull and scalp and move the auricle as a whole, and (2) intrinsic, which extend from one part of the auricle to another. Since these muscles are rudimentary, they have no particular surgical importance. In the infraprimate mammals these muscles are particularly well developed and are important in turning the auricle toward the source of a sound.

The motor innervation is by the temporal branch and the posterior auricular branch of the facial nerve. The sensory nerves are the great auricular nerve, from the cervical plexus; the auricular branch of the vagus nerve; the auriculotemporal branch of the mandibular nerve, and the lesser occipital nerve, from the cervical plexus.

The arteries of the ear are composed of the posterior auricular, from the external carotid artery; the anterior auricular, from the superficial temporal artery, and a branch from the occipital artery. The veins accompany the corresponding arteries. The lymphatics drain into the periauricular nodes.[14]

In examining the normal ear from behind, it will be noted that the concha forms a 90 degree angle with the head. The scapha likewise forms an angle of approximately 90 degrees with the concha, but this may vary as much as 10 to 15 degrees and still be within normal limits according to Young.[15] The rim of the helix is then noted to turn outward slightly. The angle between the scapha and the concha is formed by the anthelix, and in the absence or underdevelopment of the anthelix the angle may become obtuse, thereby resulting in a protrusion of the auricle away from the head. Since the angle of the anthelix more or less determines the degree of protrusion of the ear, the pathologic

14. Gray.[12] Spalteholz.[13]

15. Young, F.: The Correction of Abnormally Prominent Ears, Surg., Gynec. & Obst. **78:** 541-550, 1944.

condition would resolve itself into the type and development of the anthelix; i. e., the more obtuse the angle, the more the ear projects. The measurement of the angle formed by the head and the helix is known as the auriculomastoid angle and is usually about 30 degrees.

HISTORICAL DEVELOPMENT OF THE OPERATION

The earliest reference to the correction of protruding ears was made in 1845 by Dieffenbach,[16] who advised the removal of skin from the back of the ear, followed by the suturing of the auricular cartilage to the periosteum of the mastoid bone.

Ely,[17] of New York, is credited with being the first, in 1881, to remove both skin and cartilage for the correction of protruding ears. He excised the skin and cartilage through its entire thickness, thereby leaving a scar on the anterior surface of the auricle. Fishman and Fishman [18] advised a similar procedure in 1946.

In 1890 Keen,[19] of Philadelphia, reported a method of removing a narrow, V-shaped section of cartilage from the posterior surface of the auricle after a large section of skin had been excised from this area. To avoid scarring, he was careful not to cut through the anterior surface of the skin. He expressed the opinion that a section of cartilage must be removed, for "if the skin alone is removed, the natural elasticity of the cartilage would stretch the skin in time and probably reproduce the deformity."

Monks,[20] of Boston, in 1891 described two types of operation, one in which only the skin was excised and a second in which both cartilage and skin were removed. He stated the opinion that an excision of skin from the back of the ear would suffice in most cases and is preferable to excision of cartilage, especially if a large ellipse, "equal in breadth to more than half the breadth of the back of the ear," is removed. His reason for trying to avoid excision of cartilage was that a sharp ridge and vertical fold of skin appeared on the anterior surface of the auricle in the case in which he attempted this. However, he stated the belief that if the cartilage is stiff, a condition applying principally to adults, it is necessary to excise some cartilage, thereby substituting a disfigurement for a deformity. At about the same time,

16. Dieffenbach, J. F.: Die operative Chirugie, Leipzig, F. A. Brockhaus, 1845.

17. Ely, E. T.: An Operation for Prominence of the Auricles, Arch. Otol. **10**:97-99, 1881.

18. Fishman, L. Z., and Fishman, V. P.: Plastic Surgery for Outstanding Ears: A Simple Surgical Procedure, Bull. Pract. Ophth. **16**:19-21, 1946.

19. Keen, W. W.: New Method of Operating for Relief of Deformity of Prominent Ears, Ann. Surg. **12**:49-51, 1890.

20. Monks, G. H.: Operations for Correcting the Deformity Due to Prominent Ears, Boston M. & S. J. **124**:84-86, 1891.

in 1894, Haug,[21] in Germany, reported the removal of triangular wedges of cartilage along with the excision of skin on the posterior surface of the auricle.

Joseph,[22] in 1896 and also in 1902, published a procedure similar to those described by Monks and Keen, but stated that he did so without the knowledge of these previous publications. Like Monks, he divided protruding ears into two types: the soft and the hard cartilage type. He advised that for ears of the soft cartilage type an excision of skin at the auriculomastoid angle should be sufficient, while for ears of the hard cartilage type a section of cartilage from that region would be necessary along with the excision of skin. Ballenger and Ballenger[23] advised the Joseph procedure for the correction of protruding ears.

In 1903 Morestin[24] published a method similar to that described by Joseph, except that one important advancement was made. Although he excised an elliptic piece of skin and cartilage from the auriculomastoid angle as Joseph and Keen did, he stated the belief that the incision through the cartilage must extend well up to the upper and lower borders of the cartilage.

Goldstein[25] in 1908 described an operation similar to the Morestin procedure, but he sutured the cartilage to the periosteum of the mastoid.

In 1911 Kolle[26] also described a method similar to that of Morestin. He, like Morestein, stated the opinion that after the elliptic excision of cartilage at the auriculomastoid angle, a "linear incision with the scissors may be made both superiorly and inferiorly to further mobilize the springy shell of the ear, which will then be found to fall easily in place." As recently as 1947, Wolf[27] advised the Kolle procedure as "satisfactory in most instances."

At the same time as Morestin, in 1903, Gersuny[28] described a clever operation in which a skin flap is raised both from the mastoid area

21. Haug, R.: Eine einfache neue plastische Methode zur Rücklagerung hochgradig abstehender Ohrmuschelu, Deutsche med. Wchnschr. **20**:776, 1894.

22. Joseph, J.: Nasenplastik und sonstige Gesichtsplastik, Leipzig, Curt Kabitzsch, 1928; Eselsohren, Verhandl. d. Berl. med. Gesellsch. **27**:206, 1896.

23. Ballenger, W. L.; Ballenger, H. C., and Ballenger, J. J.: Diseases of the Nose, Throat and Ear, ed. 9, Philadelphia, Lea & Febiger, 1947.

24. Morestin, H.: De la reposition et du plissement cosmétiques du pavillon de l'orielle, Rev. d'orthop. **4**:289-303, 1903.

25. Goldstein, M. A.: The Cosmetic and Plastic Surgery of the Ear, Laryngoscope **18**:826-852, 1908.

26. Kolle, F. S.: Plastic and Cosmetic Surgery, New York, D. Appleton & Co., 1911.

27. Wolf, G. D.: Ear, Nose and Throat: Symptoms—Diagnosis—Treatment, Philadelphia, J. B. Lippincott Company, 1947.

28. Gersuny R.: Ueber einige kosmetische Operationen, Wien. med. Wchnschr. **53**:2253-2257, 1903.

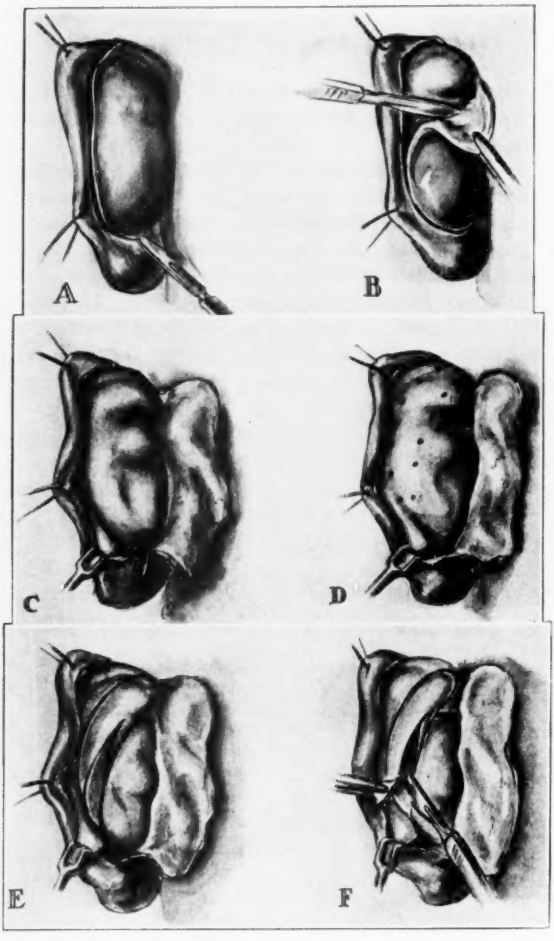

Fig. 3.—(*A*) Incision through skin and perichondrium on the posterior surface of the auricle in the region of the fossa anthelicis. (*B*) Undermining of skin and perichondrium almost to the auriculomastoid angle. (*C*) Complete exposure of the posterior surface of auricle, revealing all landmarks; undermining of the skin anteriorly to expose the cauda helicis and fissura antiragohelicina. (*D*) Outlining of the new anthelix and its superior crus. When the ear is folded back, the new anthelix is noted on the anterior surface of the auricle. The skin is then punctured through from the anterior surface with a fine needle along the outer and inner borders of the new anthelix and superior crus. Each point is marked on the posterior surface of the auricular cartilage with methylene blue to delineate the cartilage incision. No markings are necessary to outline the inferior crus, since the sulcus anthelicis transversus is usually visible. (*E*) Incision made along the markings down to the fissura antitragohelicina. An incision is also made through the sulcus anthelicis transversus to form the inferior crus. A diamond-shaped section of cartilage is then outlined, which extends up to the inferior crus. (*F*) Section of cartilage removed. The width of the excision varies with the amount of spring found in the cartilage.

and from the posterior auricular surface. The cartilage of the concha is split longitudinally into ribbons; a few narrow strips are removed, and several transverse incisions are made, leaving sections of cartilage. These sections are then sutured to the periosteum of the mastoid bone, and the skin flap is then replaced and sutured. This procedure led to many similar operations in which the cartilage is sutured to the periosteum of the mastoid.

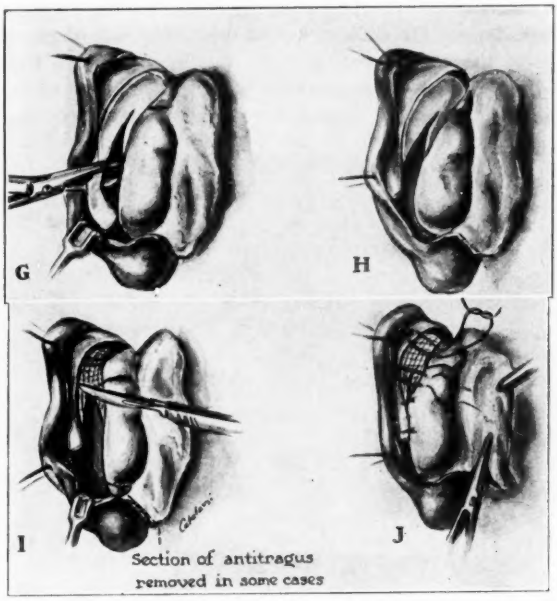

Fig. 3 (*continued*).—(*G*) Cauda helicis exposed and removed. This procedure is necessary in the majority of cases. An incision is made at the upper end of the superior crus for removal of a section of cartilage. This incision is located under the helix and is usually necessary to break completely the spring of the cartilage. (*H*) The cartilage removed. (*I*) Partial cross cutting of the remaining cartilage which forms the superior crus. These incisions are made to weaken the spring of the cartilage and must not pass completely through the cartilage. In cases in which the cartilage is soft or pliable, cross cutting is not necessary. In other cases, thinning of the cartilage by shaving is sufficient to weaken its spring. A section of antitragus is removed in most cases of extreme cupping of the lower portion of the ear. (*J*) Surgical gut fixation sutures inserted. The sutures are tied only tight enough to form a smooth anthelix and superior crus. External mattress sutures tied over cotton rolls, instead of the surgical gut sutures, have been used to good advantage in many cases. If the cartilage is soft and pliable, allowing the ear to fall back easily, no fixation sutures are necessary. Excess skin is trimmed, and black silk sutures are used to close the incision.

Following the method of Gersuny, Payr [29] in 1906 used a cross-shaped, pedunculated strip of cartilage and perichondrium from the entire width of the auricle and passed it under a loop of periosteum in the mastoid region. The results of this procedure were invariably poor, according to Davis,[30] who had tried the method. Ruttin [31] in 1910 followed a similar plan, but used a strip of fascia lata attached to the perichondrium at the inner edge of the helix and anchored it to the bridge of periosteum over the mastoid area. Luthi [32] in 1929 described a similar operation. In 1928 Alexander [33] advised a modification of these procedures. He made a longitudinal incision through skin and cartilage in the conchal region. The cartilage was then overlapped and held in position with surgical gut sutures between the cartilage and the postauricular fascia. The skin on the anterior aspect of the ear was undermined for 8 mm., evidently to eliminate the wrinkling of the skin on the anterior surface.

As late as 1935 Demel [34] still advised the removal of a large section of conchal cartilage, to be turned on itself and pedicled beneath a bridge of periosteum over the mastoid region.

In 1937, Eitner [35] recommended an operation similar to the Joseph and Morestin procedures, but tried to eliminate the wrinkling of the skin on the anterior surface by excising two triangles of cartilage with a common base from the region of the concha. The skin and perichondrium were incised horizontally and then undermined before the excision of the cartilage.

None of the procedures described can possibly result in a normal-appearing ear. The scapha projects abnormally, while the ear appears flattened against the head. On a lateral view, which is the most critical view of the ear by which to determine a good correction, the concha appears deformed in many instances, since frequently sharp ridges and excess wrinkling of the skin are noted, along with absence of the anthelix fold.

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29. Payr, E.: Plastische Operationen an den Ohren (Stellungsverbesserung, Verkleinerung), Arch. f. klin. Chir. **78**:918-928, 1906.

30. Davis, J. S.: Plastic Surgery: Its Principles and Practice, Philadelphia, P. Blakiston's Son & Co., 1919.

31. Ruttin, E.: Eine Methode zur Korrektur abstehender Ohren, Monatsschr. f. Ohrenh. **44**:196, 1910.

32. Luthi, A.: Eine einfache zuverlässige gut dosierbare Methode zur Korrectur abstehender Ohren, Schweiz. med. Wchnschr. **10**:1268, 1929.

33. Alexander, G.: Zur plastischen Korrektur abstehender Ohrmuscheln, Wien. klin. Wchnschr. **41**:1217-1218, 1928.

34. Demel, R.: Erfolge der Beseitigung der abstehenden Ohren, Wien. klin. Wchnschr. **48**:1185, 1935.

35. Eitner, E.: Eine einfache Methode zur Korrektur abstehender Ohren, Wien. klin. Wchnschr. **50**:1206, 1937.

The basic concept for the newer type of otoplasty was first described by Luckett,[36] who in 1910 pointed out that the reason for protruding ears was the absence or underdevelopment of the anthelix. If a new anthelix were made, the prominence of the ear could be overcome; furthermore, on a lateral view a more normal-appearing ear would result without the distortions of the concha and absence of the normal anthelix fold. Luckett's operation consisted of the removal of a crescentic area of cartilage and skin from the posterior surface of the auricle over the line of the proposed anthelix; the edges of cartilage were everted by the use of a Lembert suture, thereby forming a new anthelix.

Davis,[30] in 1919 and again in 1937,[8] emphasized the basic concepts of Luckett; they also served as the basis for Barsky's[37] operation in

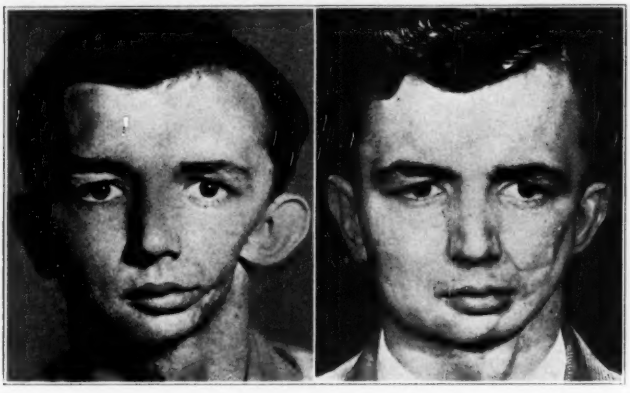

Fig. 4.—Unilateral protrusion of the ear with associated mandibular deformity. Protrusion of the ear was corrected by the method described, and the facial deformity, by a thoracicoepigastric pedicle skin graft and an iliac bone graft to the mandible.

1938. Davis and Kitlowski in 1937,[8] published a method they had been using for correction of protruding ears, based on the concepts of Luckett. They outlined the new anthelix along the inferior (anterior) crus with brilliant green introduced by needle through perforations in the skin with a needle from the anterior surface of the auricle. The skin on the posterior surface of the auricle which had been removed both from the conchal and from the mastoid region was discarded. The cartilage was split along the previously marked line and a section

36. Luckett, W. H.: A New Operation for Prominent Ears Based on the Anatomical Deformity, Surg., Gynec. & Obst. **10**:635-637, 1910.

37. Barsky, A. J.: Plastic Surgery, Philadelphia, W. B. Saunders Company, 1938.

removed. The authors emphasized that the cartilage must be split through the entire length of the proposed anthelix to break its elasticity. Surgical gut sutures closed the perichondrium, and the edges were everted to form the new anthelix.

The objection to the Davis-Kitlowski procedure is that the normal postauricular sulcus is obliterated by the excessive removal of skin from the posterior surface of the auricle and from the mastoid region. Also, in the removal of cartilage only from the inferior crus, the superior

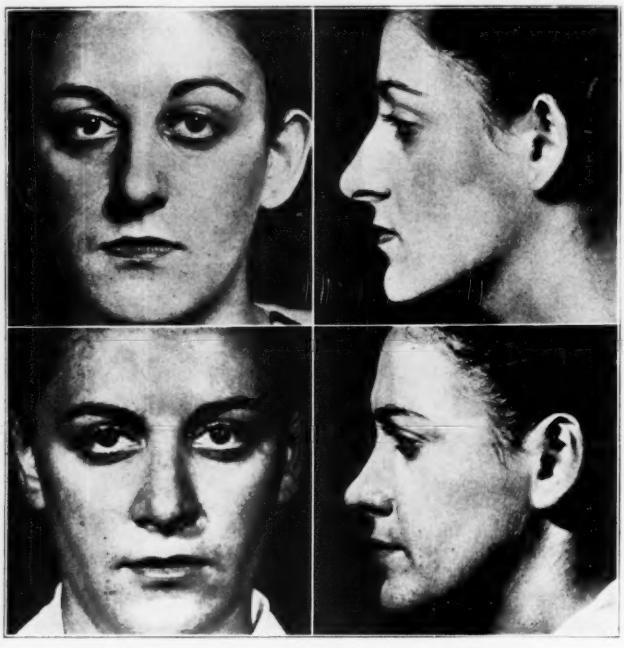

Fig. 5.—Protruding ears corrected by the standard method of excision of cartilage. Note that the corrected ears are properly placed in relation to the head, but in the lateral view a sharp ridge is present in the region of the superior crus. This gives the ear an unnatural appearance. (A rhinoplasty was performed at the same time that the ears were corrected.)

crus may appear flat, and at times the ear will appear to lop forward at its upper pole.

The Davis-Kitlowski procedure led to a modification by New and Erich[38] in 1940, who used mattress sutures externally to help hold

38. New, G. B., and Erich, J. B.: Protruding Ears: A Method of Plastic Correction, Am. J. Surg. **48**:385-390, 1940.

the form of the new anthelix. They also excised cartilage from the inferior crus, but at times felt it necessary to remove a wedge of cartilage from the superior (posterior) crus to prevent the upper pole of the ear from falling forward. New and Erich cited Webster as stating that he had satisfactory results without incising the cartilage, merely shaving the cartilage in the region of the anthelix. New and Erich, however, stated the belief that the ear can be folded back with greater ease if a narrow strip of cartilage is excised.

Weaver,[39] in 1947, described the use of the New and Erich procedure and stated that he had found it a highly satisfactory method. His experience was based on 8 cases.

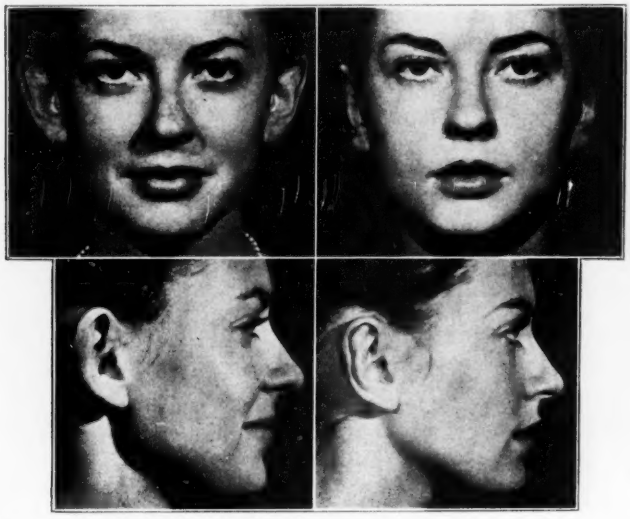

Fig. 6.—Bilateral protrusion of the ears, most marked at the upper pole. The angle of the anthelix is obtuse, accounting for the protrusion of the ear. The normal angle of the anthelix is from 80 to 100 degrees. The formation of a normal anthelix by the method described, with very little sacrifice of skin from the posterior region of the auricle, results in an ear which satisfactorily simulates the normal.

Young [15] in 1944 described a method similar to but an improvement on the Davis-Kitlowski procedure. He stated the opinion that if the section of cartilage forming the new anthelix were removed correctly the scapha could be slipped over the concha, and thereby no external mattress or internal surgical gut sutures would be needed to hold the new fold of the anthelix. The excision of cartilage was made

39. Weaver, D. F.: Correction of Prominent Ears, Arch. Otolaryng. **45**:205-208 (Feb.) 1947.

in the region of the superior (posterior) crus rather than in the inferior crus, though Young expressed the belief that an incision is necessary in the inferior crus at times. No skin, other than the excess, is sacrificed from the postauricular sulcus.

Young's procedure is a decided improvement over the Davis-Kitlowski method, for two reasons: 1. The main excision of cartilage is from the superior crus; the ear thus has a better appearance. 2. No skin is sacrificed from the postauricular sulcus; thereby the ear is prevented from appearing too close to the head. The objections to Young's method are that a sharp ridge is produced in the region of the superior crus (fig. 5) and no provision is made for extreme cupping or protrusion of the lower pole of the ear.

Fig. 7.—Marked protrusion and cupping of the ears corrected by the method described. The preoperative photographs show complete absence of the anthelix and its superior crus. The inferior crus is present but not well defined. It was necessary to remove a section of antitragus after the removal of the cauda helicis in order to set the lower portion of the ear back to its proper position.

Barsky [87] in 1938 presented an excellent operation, based also on Luckett's principles, in which he excised a narrow triangular section of cartilage in the region of the proposed new anthelix. The base of the triangle was about 1/16 to 1/8 inch (0.16 to 0.32 cm.) in width, and the apex came to a point at the perichondrium on the anterior surface. Two curved incisions were made parallel to this one on either side, and the cartilage was thinned by being shaved with a knife. Surgical gut sutures were inserted through the perichondrium to unite the two incisions and thereby form an anthelix. Barsky stated the belief that it might be necessary to excise a small ellipse of cartilage from the base of the concha.

In 1947 McEvitt[40] described a method similar to that used by Young. He advised the excision of cartilage at the anthelix and its crura, and in some cases the placement of parallel incisions further to break the spring of the cartilage. He emphasized the necessity in some cases of excising part of the antitragus and crosscutting the remaining portion, a detail which most reports fail to mention in correction of the extremely cupped type of ears. He stated that in correcting some of the simpler types of protruding ears he has lately tended to remove less and less cartilage, and at times none.

MacCollum,[41] in 1938; Coe,[42] in 1942; Seeley,[43] in 1946, and Seltzer,[44] in 1947, described operations similar to the Luckett, Young and Davis-Kitlowski methods, except that they varied the excision of the cartilage.

Fig. 8.—Protruding ears corrected by the method described. The corrected ears present a normal contour of the anthelix and superior crus in lateral view and a normal auriculomastoid angle in the posterior view.

Pierce, Klabunde and Bergeron[45] reported a procedure for correction of protruding ears by making eight to ten incisions almost

40. McEvitt, G.: The Problem of the Protruding Ear, Plast. & Reconstruct. Surg. **2**:481-496, 1947.

41. MacCollum, D. W.: The Lop Ear, J. A. M. A. **110**:1427-1430 (April 30) 1938.

42. Coe, H. E.: Correction of Lop Ears, Northwest Med. **41**: 126, 1942.

43. Seeley, R. C.: Correction of the Congenital Protruding Ear, Am. J. Surg. **72**:12-15, 1946.

44. Seltzer, A. P.: The Importance of Correcting Outstanding Ears, Ann. Otol., Rhin. & Laryng. **58**:1012-1020, 1947.

45. Pierce, G. W.; Klabunde, E. H., and Bergeron, V. L.: Useful Procedures in Plastic Surgery, Plast. & Reconstruct. Surg. **2**:358-361, 1947.

through the cartilage. They stated the belief that the older operations leave a prominent ridge on the anterior surface of the auricle and that this new method gives an accurate representation of the normal fold. Five surgical gut sutures are used to hold back the anthelix fold and the ear. Excess skin is removed and the remaining skin is sutured with dermalon.® This procedure, with Barsky's method, is a significant advance over the older procedures in which sections of cartilage are removed, for these methods allow for a smooth, and not a sharp, superior crus of the anthelix, thus simulating the normal ear. However, in my experience, these procedures are applicable only to the very slight, or simple, type of protrusion.

The fact that so many different procedures have been described for the correction of protruding ears leaves the impression that no one ideal method has been found. At times it is necessary in certain types of ears to use a variety of methods, since no one procedure will suffice for the entire correction. However, the method to be described, with the variations which are outlined for different types of protrusions, quite accurately reproduces the normal auricle with almost uniform success (figs. 4, 6, 7 and 8).

OPERATIVE TECHNIC

The hair is shaved for a distance of 1 inch (2.5 cm.) from the ear and is held away from the operative field with a head towel and strips of adhesive tape. The ears are thoroughly washed with soap and water, followed by application of an antiseptic solution. With the patient lying on his back, the head is draped, and only one ear is exposed at a time. A comparison of the two ears is usually unnecessary during the operation, since one can judge fairly accurately when the ears are evenly placed to the head.

Children are anesthetized by the endotracheal method with ether or gas; adults, by local infiltration of procaine hydrochloride, 1 per cent, with epinephrine hydrochloride, 1:5,000, after preoperative analgesia with morphine and barbiturates. A complete encirclement of the ear is made with applications of the procaine, and additional solution is infiltrated on the posterior surface and on the anterior surface of the anthelix, the injections in the latter two regions being primarily to facilitate the dissection and for hemostasis.

An incision through the skin and perichondrium is made on the posterior surface of the auricle in the region of the margin between the scapha and the concha, i. e., in the fossa anthelicis, and is extended at the superior portion to the junction of the ear and the temporal region, while at the inferior portion it is extended down to the base of the antitragus, close to the mastoid region. The skin and perichondrium are then undermined laterally almost to the auriculomastoid angle and to the beginning rim of the helix, thereby exposing the whole posterior surface of the auricular cartilage and all its landmarks (fig. 3 *A*, *B* and *C*).

The following landmarks are exposed on the posterior surface of the auricle: (1) the entire region of the concha and scapha, which is necessary to the outlining of the new anthelix and its superior crus; (2) the sulcus antihelicis transversus, which corresponds to the inferior crus of the anthelix; (3) the fissura antitragohelicini, which separates the cauda helicis from the antitragus; (4) the cauda

helicis in its entirety; (5) the conchal furrow or fissure formed by the portion of the helix which divides the concha into cymba and cavum, known as the sulcus cruris helicis, and (6), in some cases of cupped protruding ears, the antitragus (fig. 2).

If the ear is folded back against the head, the new anthelix is noted on the anterior surface of the auricle. The skin and cartilage are then punctured through from the anterior surface with a fine needle, and each point is marked on the posterior surface of the auricular cartilage with methylene blue to outline the incision in the cartilage. The double row of marks resulting from this maneuver are so placed that they completely delineate the posterior and anterior borders of the anthelix and its superior crus. At the upper end, the markings converge to a curved apex in the region of the superior crus of the anthelix as it merges with the helix. The inferior border comes to a point at the fissura antitragohelicina (fig. 3*D*).

Two incisions are made through the cartilage, one along each of the markings (fig. 3*E*). A section of cartilage is then removed from the anthelix. This excision starts at the fissura antitragohelicina and continues to an apex at the sulcus anthelicis transversus, so that a diamond-shaped piece of cartilage is removed (fig. 3*F*). The size of the excision of cartilage depends on the amount of spring which must be overcome and ranges at its widest point from 6 to 9 mm. The skin is then undermined laterally for a few millimeters on the anterior surface of the auricle. No cartilage is removed from the superior crus.

The sulcus anthelicis transversus, which corresponds to the inferior crus, is easily located and is then incised completely through. The incision extends into the previously incised cartilage of the anthelix. It will break the spring of the inferior crus, and rarely is the removal of a wedge or cross cutting of this region necessary (fig. 3*E*).

The cauda helicis, which has already been exposed, is then completely removed (fig. 3*G* and *H*) unless the deformity is completely confined to the upper portion of the ear, in which case removal may not be necessary. However, in my experience it has been necessary in almost all cases either to remove or to break the spring of the cauda helicis by cross cutting, for in most cases there are prominence and cupping of the lower portion of the ear with a projection upward of the ear lobe. Since removal of the cauda helicis does not cause any distortion of the auricle on the anterior surface, it has proved advantageous in most cases to remove it.

The superior crus is then cross cut by incisions, which do not completely pass through the cartilage (fig. 3*I*). The purpose in incomplete cross cutting of the cartilage at the superior crus is to weaken the spring of the cartilage and thereby help to give it a rounded contour, since normally the superior crus of the anthelix presents a smooth, rounded surface. This rounded contour becomes evident after the sutures are inserted. Since the lower portion of the anthelix usually presents a sharper edge, it is permissible to remove a section of cartilage from that region. When cartilage is removed from the superior crus, it leaves a very sharp ridge on the anterior surface of the auricle, which is unnatural in appearance (fig. 5).

In many cases it is not necessary to cross cut the cartilage, especially in the ears of children, in which the cartilage is soft and pliable and will bend into position after the insertion of the fixation sutures. In other cases thinning down of the cartilage by shaving is sufficient to weaken its spring.

A small transverse section of cartilage is now removed from the upper end of the superior crus, at its junction with the helix (fig. 3*G* and *H*). This is necessary to break completely the spring of the anthelix and superior crus from its attachment to the cartilage of the helix. If this section of cartilage is removed

close to the helix, no visible fold or defect will be seen on the anterior surface of the superior crus, since it is hidden under the rim of the helix.

In cases in which severe cupping and projection of the ear lobe and lower portion of the ear have not been completely corrected by excision of the cauda helicis, the antitragus is exposed and cross cut, or a strip of cartilage is removed from the under surface of the antitragus. Removal of a section of antitragus from its under surface leaves no deformity on the anterior surface of the auricle (fig. 3*I*).

In some instances, when the concha itself is also involved in the protrusion of the ear, it may be necessary to cross cut or to shave a section of cartilage at the fissure which is present in the concha. This fissure, which is formed by a continuation of the helix, is the sulcus cruris helicis, and it separates the concha into two portions, the cymba and cavum. Many normal ears have a prominent elevation on the anterior surface in this region; therefore cross cutting or shaving of the cartilage here will not cause an abnormal fold.

Three or four surgical gut (mattress type) sutures are inserted through the perichondrium along the margins of the original incisions in the cartilage and are tied just tightly enough to form the new anthelix fold (fig. 3*J*). In many cases external mattress type sutures tied over cotton rolls, as suggested by New and Erich,[88] were used instead of the internal surgical gut fixation sutures. In most cases the sutures proved to have a definite advantage over the internal surgical gut fixation sutures in formation of the anthelix and superior crus. In other cases, in which the cartilage is soft and pliable, or in which the protrusion is not too pronounced, no internal or external fixation sutures are necessary.

A section of the excess skin is removed from the posterior surface of the auricle (fig. 3*J*), and the margins of the skin are closed with interrupted fine black silk sutures. Sterile wet cotton or rubber sponges are placed in the convolutions of the ear on the anterior surface, and a fairly snug pressure bandage is applied. The bandage is left untouched for six days, after which the ears are inspected and rebandaged; if external mattress sutures were used, they are removed at this time. On the ninth day the skin sutures are removed, and a light mastoid type bandage is reapplied for a period of four or five days. The patient is then instructed to wear a tight-fitting silk stocking cap at night for one week.

CONCLUSION

With the method described, a protruding ear can be properly reconstructed to simulate the normal. The anthelix fold is reformed so that it has normal contours. No sharp ridges or projections are formed, such as are usually present when cartilage is removed from the superior crus. The ear is set back to the head at the proper angle, and the postauricular sulcus is not obliterated, as in the procedures in which large sections of skin over the mastoid area and the auricle are removed. The concha appears normal, and there is no wrinkling of skin on the anterior surface of the auricle. The whole lower pole and lobe of the ear are set back by removal or cross cutting of the cauda helicis, and, when necessary, by removal of a section of cartilage from the under surface of the antitragus. By the wide exposure of the posterior surface of the auricle, all landmarks are readily identified for correction of the deformity.

30 North Michigan Avenue.

ANESTHESIA IN PERORAL ENDOSCOPY

Especially General Anesthesia in Esophagoscopy and Gastroscopy

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THE MAJORITY of peroral endoscopic examinations can be made with local anesthesia or without anesthesia without a great deal of discomfort to the patient and with satisfactory operative results. Use of local anesthetic agents is attended with a minimal amount of risk, but alarming and sometimes fatal reactions may follow their application.

The comparative risk involved in local and general anesthesia is debatable, and the choice of an anesthetic agent cannot be made on the basis of risk alone. Except under unusual conditions and for solution of especially troublesome problems, we have found that bronchoscopic examination should be made in small children without anesthesia and in adults with local anesthesia.

Employment of sedatives preoperatively is usually advisable in case of children. In case of adults, administration of barbiturates preoperatively lessens the likelihood of reaction to local anesthetics, but it is not desirable to use so much sedation that the patient is rendered uncooperative.

There are many reasons for recommending that bronchoscopic examination be done without anesthesia or with local anesthesia. Continuous inhalation anesthesia is difficult to administer if examination is prolonged, and varying degrees of relaxation are a source of annoyance to the examiner. Regulation of the cough reflex is impossible in a patient under inhalation anesthesia, and much valuable information is unobtainable at bronchoscopic investigation unless cough is voluntarily controlled or produced. When the cough reflex is reduced or abolished, excessive secretion or blood is usually more difficult to evacuate from the tracheobronchial tree and may lead to pulmonary complications if recovery from anesthesia is prolonged. Intravenous anesthesia is objectionable in bronchoscopic study for the reasons that make inhalation anesthesia undesirable. In addition, respiration is greatly depressed by the agent and, because of inhibitory reflexes, may be suspended

entirely for a long period following introduction of the bronchoscope. In such circumstances the bronchi are frequently observed only in a state of extreme expiration, with the lumens greatly reduced in size.

In contrast to bronchoscopic study, we have found that esophagoscopic and gastroscopic investigations can be performed more easily, safely and comfortably with the patient under some type of general anesthesia, supplemented in most cases by introduction of an intratracheal tube.

Introduction of an esophagoscope into the esophagus of a child less than 3 years of age without anesthesia or under inhalation anesthesia results in pressure on the trachea and larynx, which, combined with associated spasm of the vocal cords, is usually sufficient to interfere with breathing. If examination requires more than a few moments, respiration may cease and the tube must be withdrawn and respiratory function reestablished. When one is pressed for time in such instances, hurried instrumentation may add to the risk of the operative procedure.

When prolonged investigation is anticipated, we recommend introduction of an intratracheal tube, which obviates respiratory difficulty during examination and permits continuous, even anesthesia. This procedure is particularly desirable when removing an open safety pin from the esophagus of a small child.

Children are put to sleep with vinyl ether U. S. P. (vinethene®) and ether by the open drop method, and after intratracheal intubation anesthesia is maintained according to Ayre's technic, which eliminates resistance and retention of carbon dioxide. Although some evidence of laryngeal obstruction following bronchoscopic manipulation might be expected to develop in a relatively large percentage of very young children, this complication has not been found to occur after intubation with a firm rubber or plastic endotracheal tube.

In adults, intravenous anesthesia with thiopental sodium U. S. P. (pentothal sodium®) combined with curare is applicable in most cases. An important adjunct in the technic is to apply a local anesthetic in the larynx and to instill a small amount of the anesthetic drug into the trachea. This local, preliminary anesthesia will obtund many reflexes and reduce the amount of thiopental required. An endotracheal tube is then introduced for administration of oxygen. In this way respiration may be completely controlled, and esophagoscopic or gastroscopic manipulation may be performed with ease for the examiner and the patient. The relaxation of the esophagus thus obtained is particularly desirable in removal of large foreign bodies when tenseness of the patient under local anesthesia would increase the likelihood of trauma.

Employment of intratracheal intubation is especially desirable in complete obstruction of the esophagus with retention of secretion, when

ordinary inhalation anesthesia is contraindicated because of the danger of aspiration into the tracheobronchial tree. Intratracheal intubation provides an ideal method for removal of a bolus of meat from the esophagus, since this procedure usually requires prolonged instrumentation and is, therefore, especially uncomfortable for the patient under local anesthesia.

Gastroscopic investigation is usually performed with local anesthesia, but many examinations are unsatisfactory because of distress to the patient and inability to maintain inflation of the stomach with air. Intravenous use of thiopental sodium with local anesthesia and insertion of an intratracheal tube provide opportunity for prolonged study of the stomach without distress to the patient and without the changes in appearance of the gastric mucosa that may result from emotional reactions.

Adequate anesthesia that can be prolonged indefinitely provides an ideal situation for teaching, in that any number of students may observe lesions in the stomach and esophagus without compromising the welfare of the patient.

In our experience, presence of an intratracheal tube does not interfere with introduction of tubes into the esophagus. Recovery from anesthesia is prompt and postoperative discomfort less than after examination with local or inhalation anesthesia.

MÉNIÈRE'S SYNDROME

Observations on Vitamin Deficiency as the Causative Factor

II. The Cochlear Disturbance

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THE FIRST PART of this communication was concerned with the acute phase of Ménière's syndrome—the vestibular disturbance or attacks of vertigo.[1] The second part deals with the cochlear disturbance—the deafness and tinnitus—which persists between the acute attacks, or the chronic phase.

In considering the acute phase it was pointed out that clinical observations of 87 consecutive patients with Ménière's syndrome showed them to have signs of chronic vitamin deficiency, often of severe degree; that the two groups into which patients with this syndrome have so far been divided by means of the histamine skin test present different predominating deficiencies, the histamine-negative group showing a predominant niacin deficiency and the histamine-positive group a predominant riboflavin deficiency; that the type of vertigo experienced by the two groups is not the same, and, finally, that there must now be recognized a third group giving an intermediate response to histamine intradermally and showing a mixed deficiency and a mixed clinical picture.

The present paper records the observations made on the clinical features of the chronic phase as regards these three groups and the vitamin deficiencies associated with them, and the results of treatment.

MATERIAL AND PROCEDURE

The material was the same as that used in part I, viz., 87 consecutive cases of the classic Ménière's syndrome, which have been investigated from the standpoint of deficiency of the three major components of the B complex (niacin, riboflavin and thiamine) during the twenty-one months from March 1946 through November 1947.

The procedure for each patient was also the same as in part I.

1. To record the response to standard doses of histamine diphosphate administered intradermally under standard conditions as described elsewhere.[2]

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1. Atkinson, M.: Ménière's Syndrome: Observations on Vitamin Deficiency as the Causative Factor; I. The Vestibular Disturbance, Arch. Otolaryng. **49**:151 (Feb.) 1949.

2. Atkinson, M.: Ménière's Syndrome: The Validity of the Intradermal Histamine Test, Ann. Otol., Rhin. & Laryng. **54**:801 (Dec.) 1945.

2. To determine the type of deafness from which each patient suffered and to record the type or types of tinnitus of which he complained.

3. To keep a chart of those symptoms generally accepted as accompanying certain vitamin deficiencies (fig. 1).

4. To make a corresponding examination, noting signs of niacin deficiency in the tongue; of riboflavin deficiency in the eyes, the lips, the skin of the face

CASE SHEET
FOR NUTRITIONAL DEFICIENCIES

VITAMIN A
- Night blindness
- Hyperkeratosis

THIAMINE
- Anorexia
- Constipation
- Loss of weight
- Paresthesias
- Muscular tenderness
- Fatigue
- Loss of tendon reflexes
- Palpitations
- Tachycardia
- Cardiac enlargement

RIBOFLAVIN
- Magenta tongue
- Cheilosis
- Angular stomatitis
- Seborrheic dermatitis
- Photophobia
- Lacrimation
- Eye strain
- Corneal vascularization

RIBOFLAVIN—*continued*
- Corneal scarring
- Pinguecula
- Vertigo, positional
- Headache

NIACIN
- Tongue
- Abdominal tenderness
- Anorexia
- Diarrhea
- Dermatitis
- Mental depression
- Mental confusion
- Loss of memory
- Headache
- Vertigo, rotational
- "Feel the cold"

ASCORBIC ACID
- Gums
- Joints
- Hemorrhages
- Weakness
- Recent infection

Fig. 1.—Case sheet for recording nutritional deficiencies.

and in the tongue; of thiamine deficiency in disturbances of function of the nervous system as shown by hyperreflexia or hyporeflexia.

5. To record by means of photographs the appearances of the organs mentioned in paragraph 4.

6. To treat each patient as far as possible with the particular fraction appropriate to his group, and with that alone in the first place, and to observe and record the results of such treatment.

The results of these investigations will be described in two sections, one relating to deafness and the other to tinnitus.

DEAFNESS

METHOD OF EXAMINATION

1. The degree of hearing impairment was determined when the patient was first seen, by an audiometric reading in a quiet room with an average noise level of 40 decibels. Additional audiograms were made from time to time as seemed advisable, by the same operator in the same room with the same audiometer.

2. The type of hearing impairment was determined by tuning fork tests. The tests used have been the Rinne test, the absolute bone conduction test and tests to determine high and low tone limits. The first two tests have been made with a C 256 fork, and the high tone limit was determined with a Galton whistle.

3. Masking. The validity of the audiometric reading and of the results of the Rinne test and the absolute bone conduction test of the affected ear was checked by masking the normal or better ear with a sound box.

4. Grouping. Cases have been divided with regard to types of hearing loss into the conventional three groups—perceptive, conductive and mixed. Criteria for the pure perceptive group were a positive Rinne test, diminished absolute bone conduction, a lowered high tone limit and a normal low tone limit. Criteria for the pure conductive group were a negative (reversed) Rinne test, normal absolute bone conduction, a raised low tone limit and a normal high tone limit. Criteria for the mixed group were either (1) a hearing loss of predominantly nerve type but with diminished air conduction and a raised low tone limit, or (2) a hearing loss of predominantly conductive type but with diminished absolute bone conduction and a lowered high tone limit.

RESULTS OF EXAMINATIONS

The results of the examinations are shown in tables 1 and 2. One hundred and twenty-three ears with impairment of hearing were found in the 87 patients investigated. The cases of hearing loss have been divided into three categories according to the patient's response to the histamine skin test, as was done in part I. In table 1 is shown the number of cases of each type of hearing loss which have fallen into that category, and table 2 shows the severity of the impairment in each category in terms of decibel loss, determined by averaging the losses for the four frequencies 256 to 2048.

1. Perceptive deafness. This type of hearing loss is generally considered to be the usual, indeed the almost exclusive, type associated with Ménière's syndrome. In this series of 123 affected ears, impairment of purely perceptive type was present in only 62 (50 per cent), the incidence being practically equally distributed proportionately among the three histamine groups. If to these are added the 33 cases of mixed deafness in which perceptive deafness predominated (see paragraph numbered 2), there are 95 cases (77 per cent) in which the type of impairment was purely or predominantly perceptive.

2. Mixed deafness. This type was found in 52 ears (43 per cent). The distribution is interesting. Half of the cases were found in the intermediate histamine group, the group showing mixed deficiency;

TABLE 1.—*Types of Deafness According to Histamine Groups*

Group	Perceptive Deafness	Mixed Deafness		Conductive Deafness	"Dead" Ears	Total
		P + C	C + P			
Vasoconstrictor Histamine — 20 cases, 24 ears Niacin deficiency	12 (50%)	10 (41%)	2 (9%) 12 (50%)	0	0	24
Intermediate Histamine ± 43 cases, 64 ears Mixed deficiency	32 (50%)	13 (20%)	13 (20%) 26 (40%)	3 (5%)	3 (5%) (1 post-op)	64
Vasodilator Histamine + 24 cases, 25 ears Riboflavin deficiency	18 (51%)	10 (29%)	4 (11%) 14 (40%)	2 (6%)	1 (3%)	35
Total (ears)	62 (50%)	33 (27%)	19 (15%) 52 (43%)	5 (4%)	4 (3%)	123

Bilateral Cases

Vasoconstrictor group	4 (20%)
Intermediate group	22 (52%)
Vasodilator group	11 (46%)

TABLE 2.—*Severity of Deafness According to Type and Histamine Group*

	Perceptive Deafness				Mixed Deafness								Conductive Deafness				"Dead" Ears	Total
					P + C				C + P									
Group	0—20	—40	—60	Over 60	0—20	—40	—60	Over 60	0—20	—40	—60	Over 60	0—20	—40	—60	Over 60		
Vasoconstrictor Histamine — 20 cases, 24 ears Niacin deficiency	1	5	1	5	0	2	5	3	0	0	2	0	0	0	0	0		
					10 (41%)				2 (9%)									
	12 (50%)				12 (50%)								0				0	24
Intermediate Histamine ± 43 cases, 64 ears Mixed deficiency	6	17	7	2	0	2	6	5	0	2	8	3	0	0	2	1		
					13 (20%)				13 (20%)									
	32 (50%)				26 (40%)								3 (5%)				3 (5%)	64
Vasodilator Histamine + 24 cases, 35 ears Riboflavin deficiency	2	11	4	1	0	0	4	6	0	1	2	1	0	0	2	0		
					10 (29%)				4 (11%)									
	18 (51%)				14 (40%)								2 (6%)				1 (3%)	35
Total	9	33	12	8	0	4	15	14	0	3	12	4	0	0	4	1		
					33 (27%)				19 (15%)									
	62 (50%)				52 (43%)								5 (4%)				4 (3%)	123

the other half were about equally divided between the vasoconstrictor and vasodilator groups. When these 52 cases were analyzed more carefully by dividing them according to two subtypes—one in which perceptive deafness predominated, and one in which conductive deafness predominated—it was found that perceptive deafness predominated in the 12 ears in the vasoconstrictor group by 10 to 2, and in the 14 ears in the vasodilator group by 10 to 4, while the 26 ears in the intermediate histamine group were equally divided, 13 to 13, as to predominant type. Thus perceptive deafness predominated in 33 cases and conductive deafness in 19 cases.

3. Conductive deafness. A pure type of conductive deafness was found in 5 ears (4 per cent). Of these, 3 were in the intermediate histamine group and 2 in the vasodilator group. (There were no cases of pure conductive deafness in the vasoconstrictor group.) If to these 5 cases of pure conductive deafness are added the cases of mixed deafness in which conductive deafness predominated, conductive deafness, in some degree, has been present in 24 ears (20 per cent)—in 2 out of 24 ears in the vasoconstrictor group (8 per cent), in 6 out of 35 ears in the vasodilator group (17 per cent) and in 16 out of 64 ears in the intermediate histamine group (25 per cent).

It thus appears that hearing loss of conductive type, or at least some element of it, occurs a good deal more frequently than is generally appreciated; that its incidence is preponderantly in the vasodilator and intermediate histamine groups, that is to say in the two groups in which riboflavin deficiency predominates, and that its incidence is small in the vasoconstrictor group in which niacin deficiency predominates.

4. "Dead" ears. There were 4 "dead" ears in the series, i. e., ears which did not respond to the cold caloric test or to the audiometric test with the good ear masked; 3 were in the intermediate group, 1 in the vasodilator group. Of these, 1 (in the intermediate group) was the result of a total section of the eighth nerve of the ear which was believed at the time to be the operative ear, though subsequently the opposite ear became severely affected, with great and rapid loss of hearing and a recurrence of attacks of vertigo. Of the other 3 ears, 1 had normal hearing before the onset of vertigo but hearing then began to deteriorate rapidly and in a few months was completely abolished. At no time has there been any neurologic evidence suggestive of an acoustic tumor and all symptoms except deafness and tinnitus have gradually subsided and disappeared under treatment. The other two experienced sudden and complete loss of hearing coincident with the first attack of vertigo, a loss which has been permanent.

5. Bilateral impairment. By definition the classic Ménière's syndrome demands that the hearing loss be unilateral or predominantly unilateral. However, bilateral, though unequal, involvement is far from uncommon—in this series of 87 cases 37 (43 per cent) showed involvement of both ears. The distribution in groups was: vasoconstrictor group, 4 (20 per cent); vasodilator group, 11 (46 per cent); intermediate group, 22 (52 per cent). In other words, bilateral involvement is considerably more common in the two groups which show marked or predominant riboflavin deficiency than in the group showing predominant niacin deficiency.

6. Absence of bone conduction. In the course of examining for absolute bone conduction as described, i. e., with a C 256 tuning fork and the opposite ear masked with a noise box, 18 cases have been observed in which hearing by bone conduction was absent in the affected ear, though hearing by air conduction was retained; 10 were cases of perceptive deafness, 8 were cases of mixed deafness predominantly perceptive. In all these the impairment was severe: in 7 there was a loss of 40 to 55 decibels, and in 11 a loss of 60 decibels or more. Nine cases belonged to the vasoconstrictor group, 7 to the intermediate histamine group and 2 to the vasodilator group.

THERAPEUTIC METHOD

The method of treatment has been the same as described in part I, i. e., at first intensively by intravenous injection together with oral administration, later by long-continued, high dosage oral administration alone. Members of each group were treated with the appropriate vitamin fraction, and so far as possible with that alone, until satisfactory evidence of its effect had been obtained. Members of the vasoconstrictor group received nicotinic acid, members of the vasodilator group riboflavin, members of the mixed group first one and then the other. However, it has not been possible to use individual fractions to the same extent that it was in dealing with the acute phase. The vestibular disturbance can usually be brought under control within a few weeks at most, given intensive treatment and adequate dosage, and it was thus possible in treating patients for the acute phase to concentrate on one fraction and observe its effect. The cochlear disturbance constituting the chronic phase is more difficult to control; improvement, when it does occur, may not be evident for some months, and it is seldom advisable to continue exhibiting one fraction alone for such long periods for fear of precipitating severe vitamin imbalance (see part I, cases 1 and 2). Nevertheless, even though in most cases all three main fractions were used after the acute phase had been brought under control, emphasis was still placed on one fraction rather than another, so that the dosage of nicotinic acid in a vasoconstrictor case or of riboflavin in a vasodilator case would be quite disproportionate to that of the other fractions.

THERAPEUTIC RESPONSE

The same 73 cases are available for reporting the results of treatment as were reported on in part I with regard to vertigo. These 73 cases comprise 101 affected ears, 21 in the vasoconstrictor group, 55

in the intermediate group and 25 in the vasodilator group. The results have been tabulated according to type of hearing loss and degree of improvement or worsening in each group (table 3). They have been classified as "Relieved" (which implies a return to normal hearing level), "Improved," "No Change" and "Worse." The amount of change in decibels was determined by averaging the changes for four frequencies—256, 512, 1024 and 2048.

The detailed results can be studied in table 3. They may be summarized as follows: 1. No change for better or worse has been the usual result of treatment in all three histamine groups and in all three types of hearing loss. 2. Relief, or return of normal hearing, was achieved in only 3 of the 101 affected ears, 1 in each histamine group. 3. Improvement of 10 decibels, the smallest change which can be

TABLE 3.—*Deafness: Results of Treatment*

Group	Relieved	Improved			No Change	Worse			Total (Ears)
		20 db	15 db	10 db		10 db	15 db	20 db	
	P M C	P M C	P M C	P M C	P M C	P M C	P M C	P M C	
Vasoconstrictor Histamine— 17 cases, 21 ears	.. 1 ..	.. 2	2 ..	1 1 ..	6 6 ..	1	1		21
	1 (5%)	6 (28%)			12 (57%)	2 (10%)			
Intermediate Histamine ± 38 cases, 55 ears	1	1 2 ..	1	7 1 ..	14 13 3	.. 3 ..	1 2 ..	4 1 1	55
	1 (2%)	12 (22%)			30 (54%)	12 (22%)			
Vasodilator Histamine + 18 cases, 25 ears	1	.. ..	1 ..	1 1 ..	14 6 ..	1			25
	1 (4%)	3 (12%)			20 (80%)	1 (4%)			
Total (ears)	2 1 ..	1 4 ..	1 3 ..	9 3 ..	34 25 3	2 3 ..	1 2 ..	5 1 1	101
	3	5	4	12	62	5	3	7	
	3 = 3%	21 = 21%			62 = 61%	15 = 15%			

regarded as significant, or more was achieved in 21 ears, the largest proportion in the vasoconstrictor, or niacin-deficient, group (28 per cent), the smallest in the vasodilator, or riboflavin-deficient, group (12 per cent). 4. Improvement or relief was thus obtained in 24 ears (24 per cent), which means that in these cases the pathologic process was reversed either completely or to a greater or lesser degree. 5. Deterioration in hearing took place in 15 ears, the great majority of these (12) being in the intermediate histamine group with mixed deficiencies, the group in which the most severe degree of chronic deficiency is found. The treatment given was evidently either insufficient, despite often heroic dosage, or incompetent to bring the pathologic process under control.

COMMENT

Type of Deafness.—A comparison of the findings with regard to the two types of deafness shows that pure perceptive deafness occurred in 50 per cent of all cases and was evenly distributed among all three

histamine groups, while mixed deafness of predominantly perceptive type occurred in smaller and more varied proportions but still in all groups and to a considerable degree. This series of cases thus conforms to the accepted pattern.

Nevertheless, deafness of conductive type played a not inconsiderable part in the hearing loss, a much greater part than is generally realized. Pure conductive deafness was found in 4 per cent of all ears only, but mixed deafness of predominantly conductive type occurred in 16 per cent. Moreover, a minor degree of conductive deafness was present in a further 27 per cent. Thus, while pure conductive deafness appeared in only a very small proportion of the ears involved, conductive deafness mixed with perceptive deafness in greater or lesser degree appeared in considerable proportion. But, and this is an interesting point, its incidence was almost exclusively in the intermediate and vasodilator histamine groups, the groups in which a deficiency of riboflavin is considerable or predominant. In the vasoconstrictor group, the group of predominantly niacin deficiency, conductive deafness in pure form did not appear at all and was predominant in only 2 of 12 ears with mixed deafness in this group.

These observations suggest that the factor responsible for the lesion causing a perceptive type of deafness in cases of Ménière's syndrome is a deficiency of niacin, while that responsible for the lesion causing a conductive type is a deficiency of riboflavin.

Furthermore, type of deafness can be correlated with type of vertigo. In part I of this communication [1] it was shown that positional vertigo was associated with riboflavin deficiency, while rotational vertigo was associated with niacin deficiency. Here it is seen that conductive deafness is associated with riboflavin deficiency, perceptive deafness with niacin deficiency. While it does not follow syllogistically that all patients with positional vertigo suffer from conductive deafness and that all patients with rotational vertigo suffer from perceptive deafness, nevertheless it is found that those patients in whom conductive deafness predominates complain predominantly or solely of positional vertigo, while those in whom perceptive deafness predominates, i. e., the large majority, complain predominantly or solely of rotational vertigo. Put in another way, rotational vertigo and perceptive deafness and tinnitus occur together, comprise the more commonly occurring triad and are associated with, and are apparently the result of, niacin deficiency, while positional vertigo when accompanied by cochlear disturbance is associated with conductive deafness and tinnitus and with riboflavin deficiency. But positional vertigo may, and often does, occur without accompanying cochlear disturbance (pseudo Ménière's disease), and it also is associated with riboflavin deficiency, as will

be seen elsewhere.[3] Hence it would seem that niacin deficiency when it affects the ear involves both cochlear and vestibular functions, that is to say, produces its effect at the periphery, probably through the autonomic nervous system. Riboflavin deficiency, on the other hand, often affects the vestibular tract without the cochlear, and it is therefore possible that in some, at least, of such cases the site of the disturbance is central. The available evidence suggests rather strongly that riboflavin deficiency can produce a disturbance either at the periphery or in the central tract and may even involve both coincidentally (see discussion under "Tinnitus," page 583 and footnote 3).

These observations and speculations are lent some support by the findings of Kuilman [4] and De Raadt,[5] two Dutch observers who worked independently of each other in prisoner of war camps in Batavia. Kuilman observed 412 cases of what he calls "camp vertigo," vertigo of variable duration occurring at intervals from weekly to many times a day in which sudden head movement would often result in severe attacks of vertigo with nystagmus and nausea or vomiting. Thirty-seven of these patients (9 per cent) were also "camp deaf," the deafness being especially for low tones. Many of them suffered from frank pellagra, beriberi and other symptoms of nutritional deficiency which confirmed the impression of a nutritional disease. He found no evidence of vestibular damage and he believed the symptoms to be due to a central disorder. Nicotinamide, which was the only individual fraction of the B complex available to him, caused some improvement, but yeast treatment was found more effective.

De Raadt reported on 160 cases of pellagra in which almost identical manifestations were observed, though in 26 per cent of his cases tinnitus and deafness were present. Here again the deafness was of conductive type. He stated his opinion that "the underlying pathology . . . is a degenerative brain stem encephalitis" and that riboflavin deficiency plays the most important part in causing the symptoms of disturbance of the central nervous system.

Severity of Deafness.—Table 2 shows that when an element of conductive deafness was present the severity of the hearing loss increased. The most severe loss was found in the 5 ears with pure conductive deafness, all of which had a loss of more than 40 decibels. The least severe loss was in ears with pure perceptive deafness; 42 of the 62 (28 per cent) had a loss of less than 40 decibels. In the

3. Atkinson, M.: Positional Vertigo: Observations on Its Causes and Control, to be published.

4. Kuilman, J.: Camp Vertigo, Nederl. tijdschr. v. geneesk. **90**:983 (Aug. 17) 1946.

5. De Raadt, O. L. E.: Pellagra in Oto-Neurology and Rhino-Laryngology, Leiden, Universitaire Pers, Leiden, The Hague, Martinus Nijhoff, 1947.

cases of mixed deafness the losses were intermediate in severity, but still of high degree, only 7 of the 52 ears (13 per cent) having a loss of less than 40 decibels. It seems, therefore, as though the factor responsible for the lesion producing a conductive type of deafness produces a more severe disturbance than that responsible for the perceptive type. If, as has been suggested, these factors are respectively a deficiency of riboflavin and a deficiency of niacin, then riboflavin deficiency appears to be the villain of the piece. When both deficiencies are present in more or less equal degree, as in the intermediate histamine group, the resulting disturbance is also severe, as would be expected—45 of the 52 ears (87 per cent) showing a loss of more than 40 decibels and 18 of these a loss of more than 60 decibels.

Bilateral Impairment.—Cases of bilateral impairment, which point to either a more severe disturbance or a higher susceptibility, have been of much greater frequency in the intermediate histamine and vasodilator groups, in which riboflavin deficiency is a major or predominant factor, than in the vasoconstrictor group. Whereas in the vasoconstrictor group only 4 of 20 cases (20 per cent) were bilateral, in the intermediate histamine group 22 of 42 cases (52 per cent) and in the vasodilator group 11 of 24 (64 per cent) had significant impairment of hearing on both sides.

"Dead" Ears.—There were 4 such ears, in which "death" had occurred after the onset of vertigo (resulting presumably from the disturbance under consideration, not from some previous infection). They indicate a disturbance of great intensity. They also were confined to the same two groups, the intermediate and the vasodilator (table 2).

The evidence is thus considerable for indicting riboflavin deficiency as responsible for a more severe disturbance than that due to niacin deficiency.

Absence of Bone Conduction.—Absence of hearing by bone conduction with retention of hearing by air conduction has been described before as occurring in some cases of Ménière's syndrome.[6] It has been observed in this series of cases only in the presence of pure or predominant niacin deficiency. So peculiar is such a phenomenon that doubt has, perhaps naturally, been cast on the validity of the observation,[7] and until now, so far as is known, no adequate explanation has been offered for it. Dr. E. P. Fowler Sr., however, in a personal communication, has provided one. He writes:

> The maximum loudness of a tuning fork by bone conduction is usually between 50 and 60 decibels, but the loudness level of a noise box is much greater. Impedance

6. Mygind, S. H., and Dederding, D.: Les syndrômes ménièriques, Paris, Presses Universitaires de France, 1934.

7. Tumarkin, A.: Letter to the Editor, J. Laryng. & Otol. **61**:297 (May) 1946.

through the skull being small, only 4 to 5 decibels, the margin between fork by air and bone conduction may be narrow, as also that between fork and noisebox by bone conduction. If, therefore, the degree of conduction deafness is 60 decibels or more, the masking noise may, by crossed bone conduction, mask out hearing by bone conduction while some small degree of hearing by air conduction for the same tuning fork is retained. In other words, if the contralateral masking sound is effective, it will be sensed also in the homolateral ear and therefore mask both ears.

Results of Treatment.—Improvement in the degree of deafness of whatever type and in whichever group has been the exception. This accords with the general experience. In all, only 24 ears have shown any degree of improvement, though 5 of these gained 20 decibels or more, and in another 3 hearing returned to a normal level. No ears with pure conductive deafness showed any improvement, but 13 ears (24 per cent) with pure perceptive deafness improved, 1 returning to normal (table 3). It has already been mentioned that the best results were obtained in the vasoconstrictor (niacin deficiency) group, the poorest in the vasodilator (riboflavin deficiency) group, which again points to the severity of the lesion produced by riboflavin deficiency. However, though improvement is the exception, it is usually possible to prevent further deterioration of hearing and to overcome the distortion of hearing of which some patients complain. When, as not infrequently is the case, the patient experiences considerable spontaneous variation in hearing level, adequate treatment can restore hearing to the highest level attained spontaneously and maintain it there. This in itself is often a considerable achievement, for the highest level may approach the normal. It has been classified, however, not as improvement but as maintenance.

Although any marked improvement of hearing is exceptional, when it occurs the result may be startling. The audiograms shown in figure 2 are examples, one from each histamine group, of what can from time to time be achieved by exhibiting the suitable vitamin fraction in high dosage. The objective results obtained in this series with this method of treatment show a slight though definite improvement over those previously published by me,[8] and the subjective improvement is often considerably greater. It must, however, be repeatedly insisted that such results, or any results in cases of Ménière's syndrome, have been achieved only by patient and persistent administration over long periods of the appropriate fraction or fractions in what by conventional standards must be regarded as extremely high dosage—nicotinic acid, 400 to 600 mg.; nicotinamide, 1.5 to 2.0 Gm.; riboflavin, 160 to 240 mg.; thiamine, 200 to 400 mg. daily.

8. Atkinson, M.: Ménière's Syndrome: Results of Treatment with Nicotinic Acid in the Vasoconstrictor Group, Arch. Otolaryng. **40**:101 (Aug.) 1944.

Reasons for Failure.—The question immediately arises why, when the hearing of a minority of patients can be so dramatically improved, the majority can expect only prevention of further deterioration, some

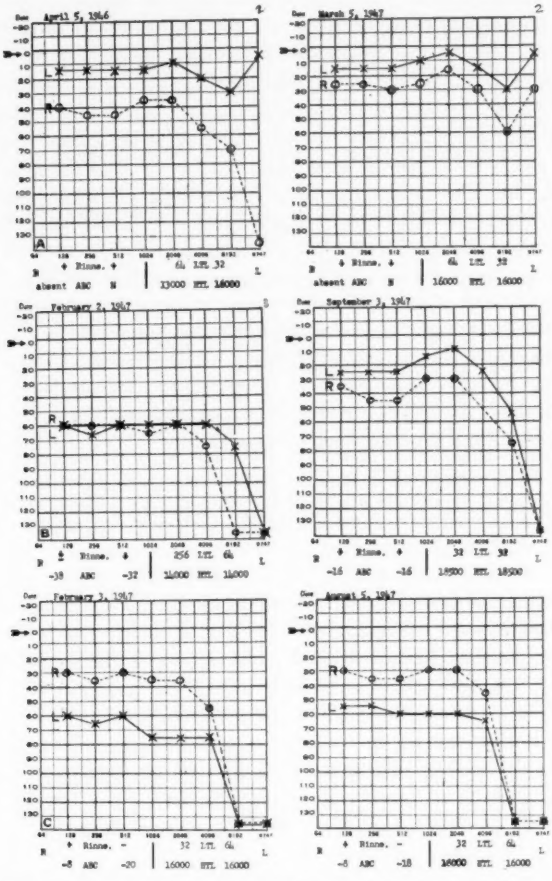

Fig. 2.—*A*, audiograms of a woman aged 23 years belonging to the vasocontrictor group. She had perceptive deafness in the right ear. The hearing of this ear improved when she was treated with nicotinic acid. *LTL* signifies low tone loss; *HTL*, high tone loss; *ABC*, absolute bone conduction. *B*, audiograms of a man aged 35 years belonging to the intermediate group. He had severe mixed deafness in both ears. His hearing was greatly improved on administration of both nicotinamide and riboflavin. *C*, audiograms of a woman aged 61 years, belonging to the vasodilator group. She had mixed deafness, preponderantly conductive, in the left ear and mild perceptive deafness in the right ear. The hearing revealed little improvement after treatment with riboflavin.

not even that. One possibility lies in insufficient dosage, despite the high doses used, a possibility to which support is lent by the marked deterioration of hearing which has taken place in a few patients who stopped taking their oral dose when the attacks of vertigo ceased, and the equally marked improvement which has occurred with very massive dosage in a few of the patients on whom such dosage has been tried. This matter is at present under investigation, but many months must elapse before any sort of conclusion can be reached—the reversal of tissue change is of necessity a slow process even if it can be accomplished.

With this advocacy of high dosage, however, must go a warning. As with drugs, so with vitamins, there is a limit to individual tolerance, which in the case of vitamins varies as a general rule inversely with the age of the patient and the duration, i. e., the chronicity, of the deficiency. The older the patient and the more long-standing and severe the deficiency, the less can he tolerate heavy dosage. It often makes such patients very uncomfortable, presumably by forcing a rusty metabolic mechanism to undue activity. However, they usually respond satisfactorily in terms of general well-being to small doses, and by such means degenerative changes can often be slowed down or even contained.

Here, then, is another reason for failure, irreversible tissue change. This is by no means always confined to elderly patients with degenerative lesions. Many younger patients have been encountered who have become severely deafened suddenly or within a day or so of the onset of vertigo and whose hearing has shown no change thereafter, despite energetic treatment. Presumably irretrievable damage has taken place. This likelihood is supported by the autopsy observations of Hallpike,[9] who has reported cochlear degeneration in 3 of 5 cases. What occurs to produce this damage is at present not known.

A third possibility is the presence of some complicating factor which has been overlooked, such as an undiscovered source of infection, concurrent disease or some emotional disturbance—the last far from uncommon. Consideration of the role played by such factors must be left for a subsequent communication.

Finally, there may be some additional and as yet unrecognized, even unknown, factor to account for some cases of failure.

9. Dix, M. R.: Hallpike, C. S., and Hood, J. D.: Observations upon the Loudness Recruitment Phenomenon, with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and VIII Nerve, Proc. Roy. Soc. Med. **41**:516 (Aug.) 1948.

TINNITUS

MATERIAL AND PROCEDURE

The material was the same 87 cases with which this investigation has been concerned throughout. The number of ears involved with tinnitus, however, was only 98, compared with 123 deaf ears. Thus 25 of the 36 patients who had bilateral hearing involvement (table 1) complained of tinnitus in one ear only.

The procedure was the same as that described in the previous section.

METHOD OF EXAMINATION

Each patient was carefully interrogated as to the sound or sounds which he heard and was asked to give as accurate a description as he could of each variety, and to indicate its severity.

An attempt was made at the beginning of this investigation to check pitch and intensity by balancing each on the audiometer. It was found impossible in most cases to do either effectively, and even when it could be done, results varied so much from one examination to another, while subjectively conditions remained much the same, that they seemed confusing rather than informative. This method of investigation was consequently abandoned, and reliance was placed solely on what each patient stated as to pitch and intensity of tinnitus and results of treatment, as well as on his description of the type of noise. It might be expected that such a subjective approach would be highly unsatisfactory, and certainly a more objective one would have been preferred. However, as it turned out, the similarity of descriptions of different varieties of tinnitus has been remarkable and the acuteness with which variations of pitch and changes of intensity were observed, sometimes by the most unexpected patients, has been a matter of surprise. As to results, the reaction of the patient in so subjective a condition is, from the clinical standpoint, perhaps as valuable as an audiometer reading of intensity.

RESULTS OF EXAMINATIONS

Typing of Tinnitus.—Very early in this investigation it began to appear that two basic types of tinnitus could be distinguished. The outstanding characteristic of one, and the more frequently encountered, was the steady continuity of the sound; that of the other was a rhythmic or surging quality, though not pulsatile or synchronous with the heart beat.

The sounds heard in the two groups have been described by patients as follows:

1. Steady sounds: (*a*) "Buzz," "hum," "escaping steam." These three frequent descriptions, the last perhaps the most usual, seem to apply to a constant tinnitus, usually of low intensity, the difference conveyed by the different descriptions being of pitch rather than of quality.

(*b*) "Whistle." From time to time the low-pitched sounds of (*a*) may rise in pitch to a high whistle, commonly likened to that on a peanut vendor's stand. This change of pitch, if persistent, often denotes a period of exacerbation and may be the prelude to an attack of vertigo. Eventually the whistle may gradually replace the steam sound largely or entirely, an indication of increasing severity of the dis-

turbance, which is further shown by an accompanying diminution of hearing.

(*c*) A "ringing or bell-like" sound. This also is a steady sound, intermediate in pitch and intensity between a buzz and a whistle. Because the sound when present is steady and continuous, it has been included in this group. Whether or not it is due to the same disturbance as those in (*a*) and (*b*) and has the same site of origin is at present undetermined. In no case in this series—indeed, in no case that I can recall—has a buzz been said to change to a ringing sound, though the change from a buzz to a whistle has often been described. Further, though "ringing in the ears" is an everyday expression (hence the term "tinnitus"), yet patients when pressed for a description of the sound do not often use the term. It may therefore be that the sound of ringing as distinct from buzzing or whistling indicates a different lesion or a different site.

2. Rhythmic sounds: Two descriptions are commonly used for this group of sounds: "like a waterfall" and "like the roar of the ocean." In both these varieties the sound is of low pitch. The descriptions seem to be applied to sounds of the same kind but of different intensity. A third description sometimes applied to the rhythmic type of tinnitus is "the hum of machinery," but here some care in interrogation is necessary; for some patients mean by this description a rhythmic sound, others a constant sound.

It must be repeated, lest misunderstanding arise, that the rhythmic type of tinnitus referred to here is not pulsatile or synchronous with the heart beat, nor is it associated with hypertension. Strictly speaking, "pulsatile tinnitus" is not tinnitus at all; it is merely the perception of an extrinsic sound, the pulse. The term "tinnitus" would be better restricted to the intrinsic type resulting from some disturbance in the auditory tract.

3. Multiple sounds: While many patients experienced one sound or one type of sound only, many others suffered at different times, or even at the same time, from both the steady and the rhythmic types. Here, then, was a third group with mixed tinnitus.

Types of Tinnitus Correlated with Type of Deafness.—These observations suggested the possibility that the three types of tinnitus—steady, rhythmic and mixed—could be correlated with the three types of deafness —perceptive, conductive and mixed. Such a correlation can in fact be made.

Consideration of table 4 shows that of the 42 ears with deafness of pure perceptive type, 37 (88 per cent) had steady tinnitus only, none had rhythmic tinnitus only, and 5 (12 per cent) had tinnitus of both types. On the other hand, of the very small group of 5 ears with

pure conductive deafness, none had steady tinnitus only, but 3 (60 per cent) had rhythmic tinnitus only, and 2 (40 per cent) had tinnitus of both types. This suggests that the steady type of tinnitus is an expression of the lesion causing a perceptive type of deafness, while the rhythmic type is an expression of the lesion causing a conductive type of deafness. The findings in the mixed deafness group are consistent with this suggestion. Steady tinnitus alone was present in 25 cases (48 per cent), rhythmic tinnitus alone in 5 cases (10 per cent) and mixed tinnitus in 21 cases (42 per cent). Reference to table 1 will show that these proportions are almost exactly those of the incidence of the different types of hearing loss.

As a result of these findings, it has seemed reasonable to designate the steady type of tinnitus as perceptive tinnitus, the rhythmic type as conductive tinnitus, and tinnitus in which both sounds are heard as mixed tinnitus, in conformity with the nomenclature of deafness.

TABLE 4.—*Tinnitus: Types Associated with Different Types of Deafness*

Types	Constant	Mixed	Rhythmic	Total	
Perceptive deafness (42 ears).....	37 (88%)	5 (12%)	0	42 ..	Cacophonous 1
Mixed deafness (51 ears).....	25 (48%)	21 (42%)	5 (10%)	51 ..	Cacophonous 5
Conductive deafness (5 ears).....	0	2 (40%)	3 (60%)	5 ..	....
Total	62 (63%)	28 (30%)	8 (7%)	98 ..	....

Unclassified Sounds.—Three other sounds, which cannot as yet be classified with any certainty, have been complained of from time to time:

1. Cacophonous tinnitus. This form of tinnitus luckily has not been common. It is a jangling medley of discordant sounds, "an orchestra playing out of tune"; it is most distressing but fortunately is usually short lived. "Indeed," said one sufferer, "I do not think it could be endured for long." It has occurred only in the most severely afflicted patients, has always been associated with other types and is probably a severe form of mixed tinnitus. There have been 6 instances in this series, 5 in patients with mixed deafness and 1 of relatively mild degree in a patient with apparently pure perceptive deafness. All the patients were having frequent severe attacks of vertigo, and in all the cacophonous tinnitus disappeared rapidly under treatment, together with the attacks of vertigo.

2. "Explosions." Sudden loud bangs in the ear have been complained of by members both of the vasoconstrictor and of the intermediate histamine groups. They have not been encountered in any member of the vasodilator group. Their origin is undetermined, but the likelihood seems to be that of some sudden vascular effect such as

a temporary release of vasospasm. They have occurred only in the more severe forms of the syndrome or during periods of exacerbation, and, like cacophonous tinnitus, are not an isolated phenomenon. They have ceased quickly once adequate treatment has been instituted.

3. Clicking sounds. Repeated clicking in the ear, often regular and long sustained, has occurred infrequently in all groups. The origin of this sound is not certain, but it is usually believed, probably correctly, to be an extrinsic sound originating in the muscles of the pharynx.

Grouping of Cases of Tinnitus According to the Results of the Histamine Skin Test.—In table 5 the numbers of cases of the three types

TABLE 5.—*Tinnitus: Types According to Histamine Groups*

Group	Perceptive Tinnitus				Mixed Tinnitus				Conductive Tinnitus				Total (Ears)
	+	2+	3+	4+	+	2+	3+	4+	+	2+	3+	4+	
Vasoconstrictor Histamine — 20 cases, 24 ears Niacin deficiency	1	6	12	0	0	0	4	0	0	0	1	0	24
	19 (80%)				4 (16%)				1 (4%)				
Intermediate Histamine ± 42 cases, 47 ears Mixed deficiency	4	5	19	1	0	4	6	6	0	0	2	0	47
	29 (62%)				16 (34%)				2 (4%)				
Vasodilator Histamine + 24 cases, 27 ears Riboflavin deficiency	2	4	7	1	0	1	2	5	3	0	2	0	27
	14 (52%)				8 (30%)				5 (18%)				
Total (ears)	7	15	38	2	0	5	12	11	3	0	5	0	98
	62 (63%)				28 (30%)				8 (7%)				

+ = soft; 2+ = medium; 3+ = loud; 4+ = distressing.

Bilateral Cases

Vasoconstrictor group	4 (20%)
Intermediate group	5 (12%)
Vasodilator group	3 (12%)

of tinnitus which have occurred in each of the three histamine groups is shown, and the degree of severity indicated by the patient has been designated as 1 plus to 4 plus as explained in the key. The "4 plus" cases are discussed more fully in a later paragraph.

It will be seen that by far the most common type to appear in pure form was the steady perceptive type. It occurred in 50 per cent or more of all ears in each group—in the vasoconstrictor group in as much as 80 per cent of 24 ears. The rhythmic, conductive type alone was not common, occurring in only 4 per cent of ears in the vasoconstrictor and intermediate groups, but in 18 per cent of 27 ears in the vasodilator group. When associated with perceptive tinnitus, as the

mixed type, however, conductive tinnitus was much more common, and its relative incidence is interesting. In the vasoconstrictor group conductive tinnitus, alone or associated, was present in only 16 per cent of ears, but in the vasodilator group it was present in 30 per cent of ears and in the intermediate group in 34 per cent. These figures follow closely those for hearing loss (see table 2), a finding which lends further support to the contention that each type of deafness has its own type of tinnitus.

To translate these findings into terms of vitamin deficiency, perceptive tinnitus, like perceptive deafness, is associated with niacin deficiency and conductive tinnitus, like conductive deafness, with riboflavin deficiency, while mixed tinnitus, like mixed deafness, is associated with a deficiency of both.

Distressing Tinnitus.—Tinnitus of this severity is luckily not common, causing as it does great mental distress, interfering with sleep and adding greatly to the sufferings of the patient. Thirteen patients have presented examples of it in this series: the 6 patients with cacophonous tinnitus already mentioned, 5 patients who described in varying terms a constant rhythmic roar which they found deeply disturbing and which was usually associated with other sounds and 2 patients who complained of a steady screaming sound, unadulterated by any other.

The distribution of these patients according to histamine grouping is interesting (table 5). There were no such patients in the vasoconstrictor group; 6 were in the vasodilator group and 7 in the intermediate histamine group. Thus all fall into one of the groups in which riboflavin deficiency is a major factor. Here is further confirmation of the observation noted in the previous section on deafness, that riboflavin deficiency produces a more severe type of disturbance than niacin deficiency.

Bilateral Tinnitus.—Bilateral tinnitus has been considerably less frequent than bilateral impairment of hearing—12 cases, compared with 37. It was present in all 4 cases of bilateral impairment of hearing in the vasoconstrictor group, but in only 5 of the 22 in the intermediate group and 3 of the 11 in the vasodilator group. It would seem, therefore, that niacin deficiency is more liable to produce tinnitus than riboflavin deficiency even though riboflavin deficiency appears to produce the more severe disturbance. This is not so paradoxic as it may sound (see "Comment").

"Dead" Ears.—In the previous section 3 ears were reported "dead" to audiometric and vestibular test yet appeared to be the operative ears, the ear on the other side being normal to test. In each case tinnitus was present. In a fourth ear, "dead" as a result of total section of the eighth nerve, tinnitus was also present. Moreover, in 3 other

cases in which the ears were "dead," reputedly as the result of an acute specific fever in childhood, tinnitus of the "dead" ear was complained of and was stated to have been present "always." This suggests either that present tests of function are not delicate enough and that these apparently "dead" ears were not in fact functionless, or that the sensory disturbance which is known as tinnitus can be displaced centrally to a higher level, so that the stimulation of the sensorium continues even though the end organ has ceased to function (see "Comment").

THERAPEUTIC METHOD AND RESPONSE

Again the same 73 cases are available for reporting results of treatment as in the previous section, but the number of ears affected with tinnitus was only 81, compared with 101 ears showing impairment of hearing (table 3). The method of treatment was that described in the previous section.

TABLE 6.—*Tinnitus: Results of Treatment*

Group	Relieved P	M	C	Total	Improved P	M	C	Total	No Change P	M	C	Total	Worse P	M	C	Total	Total (Ears)
Vasoconstrictor Histamine — 17 cases, 21 ears	3	1	0	4	11	1	1	13	3	1	0	4	0	0	0	0	21
	4 — 19%				13 — 62%				4 — 19%				0				
Intermediate Histamine ± 38 cases, 42 ears	5	1	0	6	9	5	4	18	9	4	1	14	3	1	0	4	42
	6 — 14%				18 — 42%				14 — 35%				4 — 9%				
Vasodilator Histamine + 18 cases, 18 ears	2	0	0	2	8	2	1	11	2	3	0	5	0	0	0	0	18
	2 — 11%				11 — 61%				5 — 28%				0				
Total (ears)	10	2	0		28	8	6		14	8	1		3	1	0		81
	12 — 15%				42 — 51%				23 — 29%				4 — 5%				

The results of treatment have been considerably better with regard to tinnitus than with regard to deafness, as will be seen by comparing tables 3 and 6. Whereas only 3 per cent of patients with hearing loss in all three groups have been relieved (returned to normal), 15 per cent have been completely relieved of tinnitus; whereas 21 per cent of patients have been improved as regards deafness, 51 per cent have been improved as regards tinnitus, and whereas 15 per cent of patients have deteriorated as regards hearing, only 4 per cent have deteriorated as regards tinnitus. Thus tinnitus has been more easy to control than impairment of hearing, and this applies to all groups and all types. It is worthy of note that the only group in which worsening of tinnitus occurred in spite of treatment was the intermediate group, in which mixed deficiencies of severe degree were the rule. Indeed, the results in general in that group were worse than in either of the other two. The most satisfactory results have been obtained in the vasoconstrictor (niacin-deficient) group. This conforms to the findings for vertigo and hearing loss.

It must be emphasized again here that constant, unremitting treatment, often extending over many months, may be necessary to obtain the results described. Control of tinnitus, like improvement of hearing, is uncertain and in any event seldom comes about rapidly, since it involves repair of damaged nerve tissue, notoriously a slow process. Physicians must realize this and patients be made aware of it if disappointment and disillusionment are to be avoided.

COMMENT

Site of Origin.—As has been pointed out elsewhere,[10] the auditory tract is phylogenetically a peripheral sensory tract modified to subserve the function of a special sense. Intrinsic tinnitus represents a disturbance of function of that sensory tract and may thus be regarded as auditory paresthesia, while deafness, which represents a more severe degree of that disturbance, to the point of partial or complete abolition of function, may be regarded as auditory hypesthesia or anesthesia. In other words, if this concept is valid, the symptoms known as deafness and tinnitus are evidence of a disturbance of function of a sensory mechanism, and the problem of their causation should be considered from this point of view.

This approach, however, introduces certain difficulties, having regard to current otologic concepts. The first difficulty concerns nomenclature and site of origin. Conductive, or middle ear, deafness denotes to the otologist a lesion which interferes with the "conduction" of sound waves to the inner ear and which may be situated anywhere between the external auditory canal and the articulation of the stapes in the oval window, in what may be called the external auditory apparatus. Now this external auditory apparatus, comprising the pinna, the external auditory canal, the tympanic membrane and the ossicles, is in actual fact no more than a mechanism, however complicated in structure and action, for collecting and modifying sound waves in order to transmit them at suitable intensity to the receptor end organ of Corti. Such a receptor has been called by Sherrington a distance receptor. From the physiologic point of view, therefore, the external and middle ear should be included with the cochlea as part of a receptor mechanism. If this orientation is correct, a lesion of the end organ should give rise to the same signs as those which are characteristic of a lesion of the external auditory apparatus. In other words, in the absence of evidence of a lesion of the external auditory apparatus, signs of conductive deafness implicate the organ of Corti (fig. 3).

This concept runs counter to present day otologic teaching, but it would seem that only if it is true can the fact of tinnitus associated

10. Atkinson, M.: Tinnitus Aurium: Observations on Its Nature and Control, Ann. Otol., Rhin. & Laryng. **53**:742 (Dec.) 1944.

with a lesion of the middle ear and its accompanying conductive deafness be adequately explained. For, if it is agreed that tinnitus is auditory paresthesia, then tinnitus can arise only from disturbance in auditory nerve tissue, and no part of the auditory tract enters the middle ear. But if the end organ is included with the middle ear as part of that apparatus a lesion of which causes the condition known as conductive deafness, then tinnitus associated with such deafness at once becomes

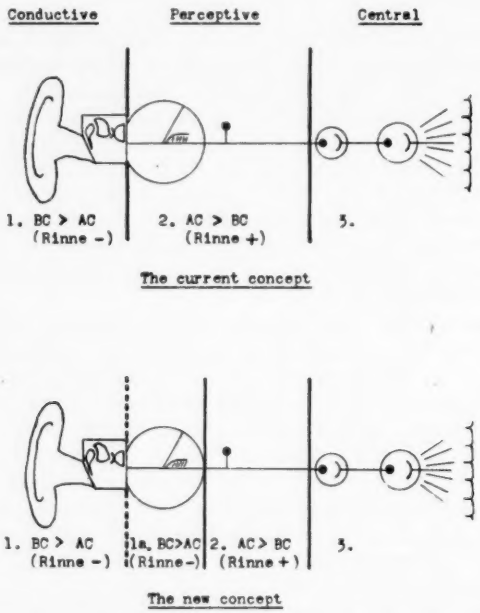

Fig. 3.—Diagrams illustrating the current concept and a new concept of the sites of lesions causing different types of deafness. *AC* stands for air conduction; *BC*, for bone conduction.

1. Middle ear deafness: *BC* is better than *AC* because the lesion interferes with the transmission of sound waves.

1 a. Cochlear deafness: *BC* is better than *AC* because the damaged cochlea responds more effectively to the powerful mechanical stimulus employed to transmit sound waves through bone than to the less powerful normal stimulus of air transmission.

2. Nerve deafness: *AC* is better than *BC*, the normal relationship, but both are reduced because a lesion of the nerve interferes with conduction of normal impulses produced by an undamaged cochlea.

3. Central deafness: A rarity not under consideration.

intelligible as arising from a cochlear disturbance. Moreover, if the preceding argument is acceptable, a reasonable explanation is at once

available for the observations reported in this paper, that tinnitus associated with conductive deafness is of a type different from that associated with perceptive deafness. The one arises in the end organ, a highly differentiated nervous structure, the other in the auditory nerve, a relatively undifferentiated nervous structure, and it might be expected that the two would be different. Further, the observation that the two types of tinnitus seem to result from different vitamin deficiencies becomes also explicable, since it is well known that deficiency of specific fractions of the B complex affects specific organs. This fact in its present reference will be discussed more fully later.

Turning now to perceptive, or nerve, deafness, this denotes to the otologist a lesion of the inner ear and/or the auditory nerve. The physiologic process, however, is that sound waves received by the the organ of Corti are converted there into electrical impulses, and these impulses are conducted along the nerve fibers to the central tracts and so to the perceptive sensorium. The type of deafness which arises from a lesion of the nerve, therefore, though called perceptive, is actually the result of an interference with conduction of nerve impulses. Moreover, inner ear deafness and nerve deafness are not to be regarded as synonymous, if this concept is valid, since physiologically the end organ belongs to the external auditory apparatus.

It has seemed necessary to discuss at some length this question of the sites of origin of the two different types of deafness, and in particular the physiologic innovation of orienting the end organ with the external auditory apparatus rather than with the auditory nerve, because only in some such way can an explanation be offered for the observations recorded in this paper. These observations show that a distinctive type of tinnitus is associated with each type of deafness, and since the two types of deafness result from lesions at different sites, the corresponding types of tinnitus presumably arise at the same sites. But if true intrinsic tinnitus can arise only in nerve tissue, then, while the explanation of intrinsic tinnitus associated with a lesion of the peripheral nerve is simple, the explanation of intrinsic tinnitus associated with deafness of middle ear, or conductive, type is impossible unless the end organ is accepted as part of the external auditory apparatus. If it is so accepted, then the rest is simple. Deafness of so-called conductive type not the result of a lesion of the middle ear or the ossicles must be due to a lesion of the end organ, is cochlear deafness, and the accompanying tinnitus arises there. Deafness of so-called perceptive type is due to a lesion of the auditory nerve trunk, and the accompanying tinnitus arises in the nerve fibrils composing it.[11]

By orienting the cochlea with the external auditory apparatus and away from the nerve trunk one is able to account for a finding which

11. Fowler, E. P.: Tinnitus Aurium in the Light of Recent Research, Ann. Otol., Rhin. & Laryng. **50**:139 (March) 1941.

was demonstrated but not discussed in the section on deafness and which is otherwise difficult to explain—that hearing loss of "nerve" type is associated predominantly with deficiency of one factor (niacin), while hearing loss of "middle ear" type is associated predominantly with deficiency of another factor (riboflavin). What is known of the clinical effects of niacin deficiency indicates that niacin is essential to the normal functioning of the autonomic nervous system, as thiamine is to the normal functioning of the voluntary nervous system. Among other things, therefore, it is concerned with the caliber of the blood vessels, a deficiency resulting in vasoconstriction. Such vasoconstriction, if it occurs in the auditory artery, will interfere with the nutrition of the nerve trunk and result in hearing loss of nerve, or "perceptive," type. The organ of Corti, being an avascular structure, will not be affected, at least not directly.

Riboflavin, on the other hand, seems to be concerned among other things with the nutrition of avascular structures, the oxidation-reduction process of its enzyme system apparently taking the place of hemoglobin in such structures. An avascular structure in which the effects of riboflavin deficiency can actually be seen is the cornea, invasion of which by new capillaries can be observed with the slit lamp,[12] while circumcorneal injection can be seen with the naked eye. These vascular changes represent an effort on the part of the body to compensate for the deficiency of riboflavin by an increase in blood supply. The organ of Corti similarly is an avascular structure, and it does not seem unreasonable to conjecture that a similar process may occur there in subjects of ariboflavinosis. And, just as in the eye dimness of vision results from corneal infiltration, so in the organ of Corti dimness of hearing may be expected to result from a similar process.

In the fact that these two deficiencies involve different structures and the fact that both deficiencies are frequently present together in considerable degree lies also an explanation of the mixed deafness and tinnitus which occur in so many cases of Ménière's syndrome. When both end organ and auditory nerve are damaged by a deficiency of riboflavin and niacin, respectively, signs of hearing loss of both types are found and tinnitus of any variety can be expected and explained.

Types of Tinnitus.—If the argument advanced is accepted, it would scarcely be cause for surprise if a lesion of so highly specialized an end organ as the cochlea produced a distorted sensory response different from, and even more severe than, that produced by a lesion of the less differentiated nerve trunk. This is what the observations recorded in the foregoing pages suggest, and the results of treatment support the suggestion. The steady type of tinnitus responds to niacin but not to riboflavin, while the rhythmic type responds to riboflavin but not to

12. Kruse, H. D.; Sydenstricker, V. P.; Sebrell, W. H., and Cleckley, H. M.: Ocular Manifestations of Ariboflavinosis, Pub. Health Rep. **55**:157 (Jan. 26) 1940.

niacin. Indeed, if nicotinic acid with its vasodilator action be given to patients with a rhythmic type of tinnitus it may increase their distress, just as nicotinic acid given to the vasodilator group will increase the frequency and severity of the attacks of vertigo.

However, if this thesis that tinnitus is a paresthesia arising in an end organ or a peripheral nerve is to be maintained, there are certain discrepancies which must be explained. Why, for instance, is tinnitus still present at times in an ear whose auditory function has been destroyed, as in the "dead" ears in this series? Why does total section of the eighth nerve only occasionally produce relief of tinnitus? The only feasible explanation would seem to be that the site of the disturbance is now no longer in the end organ or in the nerve distal to the site of section, but has been displaced centrally. This central displacement of the site of origin of a disturbance of the peripheral sensory apparatus is well known in the condition known as causalgia, in which displacement of disturbance from periphery to cord or even thalamic level is responsible for continuance of pain even after amputation, i. e., painful phantom limb.[13] Intractable tinnitus has many of the features of causalgia; for instance, there is the extreme distress caused in some cases by what to the normal person would be an inconsiderable stimulus, while tinnitus which persists in a "dead" ear is closely analogous to the pain which is felt in a phantom limb.

Effect of Treatment.—Tinnitus has been more effectively controlled than impairment of hearing—which, on the basis of this concept, is understandable. Paresthesia—in this case tinnitus—is the expression of an active, continuing process; hypesthesia or anesthesia—in this case hearing loss—is the result of that process. The result may be reversible, dependent on such factors as the severity of the process and the duration of its action; the process itself should be capable of control, at least in its early stages, provided that its cause is understood. Cessation of tinnitus indicates cessation of the active process; recurrence of tinnitus points to a recrudescence of the process and is an indication for more intensive treatment. But cessation of the active process does not automatically involve restoration of function, so that tinnitus can more often be relieved than deafness.

With regard to specific treatment, the results as regards both deafness and tinnitus have been considerably better in the niacin-deficient group than in the two groups in which riboflavin deficiency is a major factor. This conforms with the finding as regards deafness. Perhaps riboflavin deficiency is more difficult to overcome than other deficiencies.

13. Wilson, S. A. K.: Dysaesthesias and Their Neural Correlates, in Modern Problems in Neurology, New York, William Wood & Company, 1929. Livingston, W. K.: Pain Mechanisms: A Physiologic Interpretation of Causalgia and Its Related States, New York, The Macmillan Company, 1943. Henderson, W. R., and Smyth, G. E.: Phantom Limbs, J. Neurol., Neurosurg. & Psychiat. **11**:88 (May) 1948.

Certainly what is generally regarded as unusually high dosage of riboflavin (200 mg. a day) often fails to produce material improvement, though in 1 or 2 cases a still further increase of dosage has seemed to help, so that even at that level dosage may be too low. On the other hand, riboflavin deficiency may produce a more severe lesion, and thus its results may be more liable to be irreversible. This seems more probable. The evidence points to riboflavin deficiency as causing damage to the highly differentiated organ of Corti, and the more highly differentiated the structure, the more susceptible it is to alteration of internal atmosphere. It may be that to this fact is due the difficulty of reversing the disturbance of function which occurs as a result of riboflavin deficiency, whether expressed as tinnitus or deafness.

SUMMARY

Perceptive deafness has been shown to be the most common variety of hearing loss in this as in other series; nevertheless, conductive deafness in some degree has been not unusual, occurring occasionally in pure form but more usually combined with perceptive deafness, a combination termed the mixed variety of deafness.

Bilateral hearing loss, more pronounced on one side than on the other, has occurred in almost half the cases (43 per cent). Bilateral tinnitus has been present much less frequently.

Absence of bone conduction with retention of air conduction has been observed in an appreciable number of cases.

Specific types of tinnitus have been found associated with the two varieties of hearing impairment, perceptive and conductive.

Specific vitamin deficiencies have been found associated with specific types of deafness and tinnitus, niacin deficiency with the perceptive type, riboflavin deficiency with the conductive.

The suggestion has been made on physiologic grounds that the cochlea should be included with the external auditory apparatus as part of the anatomic mechanism of conductive deafness, an orientation which can explain the specific auditory effects of specific vitamin deficiencies.

The results obtained as regards deafness and tinnitus when patients were treated with the appropriate vitamin fractions have been described and discussed.

Dr. H. D. Kruse contributed advice and help in this investigation on the nutritional deficiency aspect, and Dr. Samuel Brock on the neurologic aspect.

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ACUTE DEAFNESS IN SCARLATINA

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IT IS AN old experience that occasionally a child with scarlatina may become acutely deaf and eventually deaf-mute. The acute deafness in these instances is thought to be caused by purulent panotitis or, according to the observations of Wittmaack,[1] by neuritis of the cochlear nerve. Manasse[2] was the first to emphasize that acute deafness in scarlatina may be caused by diffuse purulent labyrinthitis on one side and serous labyrinthitis on the other. In the case of Manasse[2] there was necrotic otitis media on both sides, which had caused diffuse purulent labyrinthitis on the left, and on the right, localized purulent labyrinthitis in the round window; this, in turn, had caused diffuse serous labyrinthitis. This type of serous labyrinthitis has been called "secondary" by Ruttin[3] because it originates from localized purulent labyrinthitis. It must be distinguished from "induced" diffuse serous labyrinthitis, in which there is no purulent focus in the labyrinth and in which the infection travels, rather, from the tympanic cavity through the intact windows into the inner ear.

The present case is reported to prove that acute deafness in scarlatina may be caused by common otitis media, that is, not acute necrotic otitis plus diffuse serous labyrinthitis on both sides.

REPORT OF A CASE[4]

A white girl aged 14 had a childhood history of pneumonia, measles, whooping cough, chickenpox and, at the age of 6, diphtheria. She frequently complained of trouble with the heart. On May 17, 1936, she had a slight common cold, but she was able to join her class on a trip to Salzburg on May 20. She returned to Vienna on May 24. On May 25 she complained of being fatigued, and on May 26

This article is dedicated to the memory of Hofrat Prof Dr. E. Urbantschitsch, of Vienna, in high esteem for his outstanding work in otolaryngology.

1. Wittmaack, K.: Weitere Beiträge zur Kenntnis der degenerativen Neuritis und Atrophie des Hörnerven, Ztschr f. Ohrenh. **53**:1, 1906-1907.

2. Manasse, P.: Zur Lehre von der plötzlichen Ertaubung bei Scharlachotitis und zur Kenntnis der serösen Otitis interna, Arch. f. Ohrenh. **89**:146, 1912.

3. Ruttin, E.: Die konservative und chirurgische Behandlung der Labyrinthentzündungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:104, 1927.

4. Prof. Dr. Hofrat E. Urbantschitsch performed the otologic examination and operation in this case and placed the temporal bones at my disposal for miscroscopic study.

she had fever. On May 27 she had a scarlatinal exanthem, and on May 29, slight pain in both mastoid regions. On May 31 a paracentesis was performed on both sides, releasing blood from the tympanic cavities. On the evening of this day she suddenly became deaf and could hear only the raised voice. On June 2 the temperature reached 104 F. and she was delirious; there were signs of heart failure. On June 3 the patient could not hear even the raised voice. There was no discharge from either ear.

On her admission to the hospital, on June 5, her sensorium was clear and the cranial nerves were normal. Her pulse rate was 108. She had bronchitis on the left side, strawberry tongue, eczema of the vestibulum nasi and necrosis of the pharyngeal mucosa. The lymph nodes at the angle of the mandible on the left side were swollen. The tonsils had been removed previously. The spleen was enlarged. There was scaling of the fingers, toes and trunk. No meningeal symptoms were discovered. There were 28,500 leukocytes in the blood (67 per cent polymorphonuclear cells, 28 per cent lymphocytes and 5 per cent juvenile forms) and a large number of thrombocytes. No albumin was present in the urine. On June 8 the patient was delirious at times. The right tympanic membrane was red and slightly bulged. There was a perforation in the posterior and inferior quadrant the size of a pinpoint, releasing pus with pulsating motions. There was tenderness over the right mastoid process but no edema. On the left side, the conditions were the same, but in addition there were sagging of the superior and posterior wall of the external auditory canal, edema of the tip of the mastoid and more pronounced tenderness over the mastoid. The patient was stone-deaf on the left side; on the right, she could hear only noises. There was no spontaneous nystagmus, and the caloric test revealed hypoexcitability on both sides. On account of her poor general condition, only an antrotomy was performed on both sides.

On June 10, there was a slight discharge from the right ear and more from the left. Thrush involved the mucosa of the lips and cheeks. On June 11 the temperature was 103 F. There was no dehydration, but the lymphadenitis on the left side had increased. On June 12 the temperature was decreasing, and there was moderate trismus. On June 13 the temperature rose again to 103 F. The necrosis extended toward the lateral walls of the pharynx and to the recesses of the tonsils. There were epistaxis and vomiting. The leukocyte count was 12,050, with 70 per cent polymorphonuclear cells, 18 per cent lymphocytes, 4 per cent monocytes and 8 per cent juvenile forms. On June 14 the temperature was 104 F. On June 15 the infection of the right mastoid was almost cleared, but there was discharge from the left side. The temperature was 101 F. The child felt better, but Loeffler's bacillus was cultured from the nasal secretion, and there were membranes in the right nostril. There were abducens paralysis on the left side and anesthesia of the skin of the left cheek and both corneas, more pronounced on the left side. The fundi were normal.

On June 16 the temperature was 100.8 F. The pharyngeal mucosa was red, and the tongue was coated but moist. On June 18 a small ulceration of the left cornea was noticed. The temperature fluctuated about 98.6 F. On June 22 there was a trace of pus in the right mastoid cell and a little more in the left. There were pronounced rhinolalia and a swelling at the back of the right foot. She complained of trouble in swallowing. The paralysis of the abducens and trigeminus nerves persisted. On June 26 she had no fever but had a slight paresis on the left side of the face. On July 1 there was a serous exanthem. The temperature was 99 F.; the leukocyte count was 12,150, with 60 per cent polymorphonuclear cells, 2 per cent juvenile forms, 2 per cent monocytes, 35 per cent lymphocytes, 1 per cent eosinophils and a large number of thrombocytes. On

July 2 the Pirquet reaction was negative. On July 4, there was no fever, but albumin was present in the urine. On July 6, the infection of the right mastoid was cleared, but there was a moderate amount of pus on the left side and a poor formation of granulations. On July 7 there was paralysis of the soft palate. On July 13 there was a slight discharge from the left mastoid antrum. The patient was stone deaf on the left side and on the right side had a hearing for noises only.

On July 15 an abscess had formed on the left thigh. On July 16 the paresis of the soft palate was more pronounced and there was definite rhinolalia. On July 17 there was an increase of secretion from the left mastoid. The abscess of the thigh was incised. On the morning of July 17 the child felt well, but during the day the difficulty in swallowing increased. There was paralysis of the soft palate, of the pharynx and larynx and of the left abducens nerve. In the afternoon, bronchopneumonia developed, and the patient died on July 18. Autopsy revealed that scarlatina had occurred eight weeks and diphtheria five weeks before death. The lingual tonsil was enlarged, and there was a high degree of dilatation of the heart. In the myocardium were gray foci the size of pinpoints, indicating diphtheria of the myocardium. There were bronchopneumonia of the lower lobe of the right lung and a high degree of swelling of the tracheobronchial lymph nodes, also purulent bronchitis, enlargement of the spleen and hyperemia and edema of the brain.

Macroscopic Lesions of the Temporal Bones.—In the right mastoid process there was a surgical cavity filled with granulations. There were large pneumatic cells in the squama temporalis and in the malar process. In the area of the saccus endolymphaticus the dura was split into two membranes; the inner membrane bulged, probably owing to an enlargement of the saccus endolymphaticus. The sigmoid sinus, the jugular bulb and the jugular vein were narrow, but the inferior petrosal sinus was enlarged. In the left mastoid there was a surgical cavity filled with pus. The antrum was large. The sigmoid sinus and the jugular bulb was very large, the latter extending up to the saccus endolymphaticus. The internal meatuses were normal on both sides and did not contain pus.

Microscopic Observations on the Temporal Bones.—The sections through the vestibulum and the semicircular canals on the right side had been lost.

Left Temporal Bone: The temporal bone was very well pneumatized, the cells extending to the apex of the pyramid. Some of the cells had a normal configuration, but the mucosa was extremely swollen and there was pus or serous exudate within the narrow cavity. Another part of the cells had been destroyed by the infection. The cells had been replaced by firm connective tissue which contained large cavities filled with frank pus. At the boundaries of this area were many Howship lacunas, but no osteoclasts. At other sites there was formation of new bone at the boundaries of the infected area. This area of destruction occupied the apex, extended into the floor and the roof of the internal meatus and encircled the canal of the internal meatus. Lateralward, toward the internal ear, the area became smaller and involved the periosteal layer of the bony capsule of the inner ear (fig. 1). The endochondral layer remained almost normal. The area extended above the cochlea and, mesial to the endolymphatic duct, toward the epitympanum and the mastoid, where it was continued by pneumatic cells filled with pus and swollen mucosa (fig. 2). This area did not reach the dura and did not extend into the internal ear, the internal meatus, the bulb of the jugular vein or the endolymphatic duct. But there were several small fistulas leading into the bony carotid canal (fig. 1). However, these fistulas were filled with connective tissue. The jugular bulb and the soft tissue of the carotid canal were normal.

The internal ear was poorly preserved. In the internal meatus the nerves were normal and there was no pus. In the modiolus the capillaries were enlarged and there was periosteal edema in the nerve channels. The number of spiral nerves and cells of the spiral ganglion was apparently not diminished except in the lower portion of the basilar coil, where a moderate decrease in number of the ganglion cells was discovered. Within the cochlea there was no pus, but a small amount of serous exudate, which stained with eosin, filled both the perilymphatic and the endolymphatic space (figs. 1, 2 and 3). Reissner's membrane

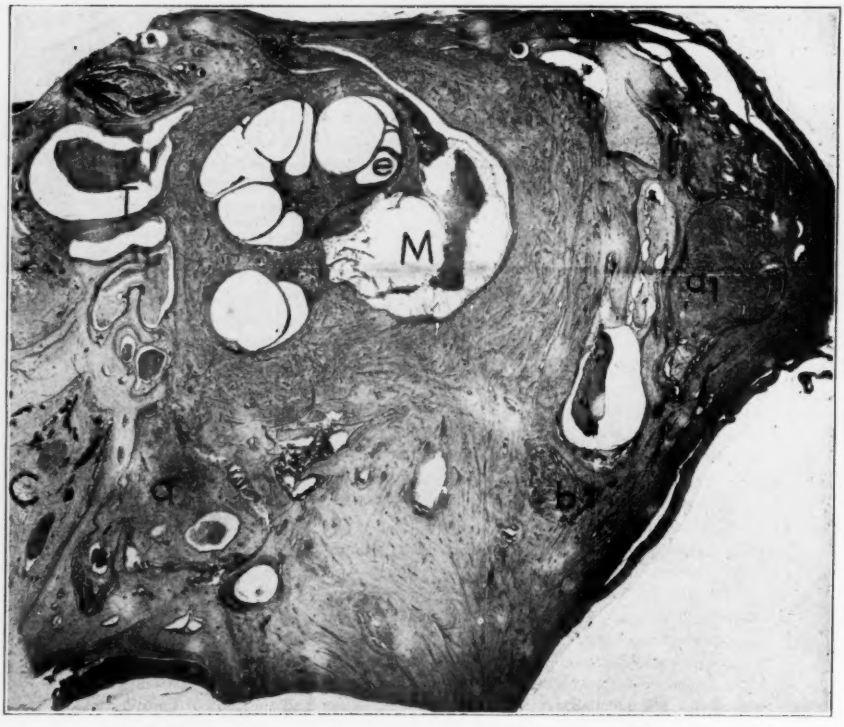

FIG. 1.—Section through the cochlea, tube (*T*), internal meatus (*M*) and carotid artery (*C*), on the right side. In this figure, *a* and a_1 indicate healing of osteitis of the petrous bone with formation of connective tissue (remnants of pneumatic cells in these areas); *b* shows a pneumatic cell of the petrous bone filled with newly formed bone, and *e*, serofibrinous exudate in the scala tympani of the basilar portion.

was extremely depressed, reducing the size of the lumen of the cochlear duct to a high degree, particularly in the superior portion of the basilar and the middle coil. In this area the cochlear duct formed a slit, filled with serous exudate. The crista spiralis, Corti's membrane and Corti's organ were compressed, but the

stria vascularis was normal. In the other coils, Corti's organ showed pronounced postmortem changes. There was serous exudate in the utricle and in the superior semicircular canal (fig. 3). The macula and the cristae were apparently normal but showed postmortem changes. The cochlear aqueduct was patent.

The mucosa of the tube and the middle ear showed massive inflammatory edema (fig. 3). Within the edematous mucosa were the ossicles, the boundaries of which showed numerous Howship lacunas. The epithelium of the mucosa was columnar and was absent only in the area in which operation had been performed. The recess of the oval window was entirely filled with the edematous mucosa. The stapes was dislocated toward the tympanic cavity (fig. 3); its boundaries were

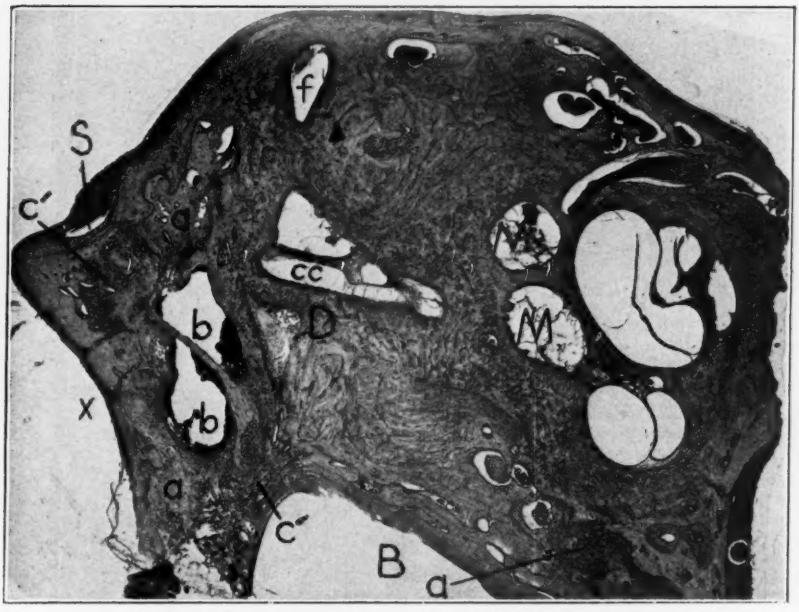

Fig. 2.—Section through the cochlea, carotid canal (*C*), internal meatus (*M*), crus commune (*cc*), frontal semicircular canal (*f*) and bulb of the jugular vein (*B*), on the left side. *D* indicates ductus endolymphaticus, presented in figures 4 and 5 in higher magnification; *S*, superior petrosal sinus, showing purulent thrombophlebitis; *a*, formation of connective tissue subsequent to osteitis of the petrous bone; *b*, frank pus within pneumatic cells, and *c'*, newly formed bones subsequent to ostetitis of the petrous bone. In *x* the osteitis had reached the dura of the posterior cranial fossa.

irregular . The frame of the oval window was normal as was the annular ligament. The floor of the bony canal of the facial nerve, above the oval window, showed an erosion which was filled with connective tissue; this erosion did not extend into the facial canal. The recess of the round window was narrow, owing to the swollen mucosa, the edema of which was not as evident as that in the oval window. The membrane of the round window was normal and bulged slightly

Fig. 3.—Section through the oval window, round window (*r*), vestibular portion of cochlea, utricle (*u*), frontal semicircular canal (*f*), crus commune (*cc*) and sagittal semicircular canal (*s*), on the left side. *M* indicates tympanal mucosa, markedly swollen and filling the recesses of both windows; *m*, macula utriculi; *c*, crista frontalis; *st*, stapes, slightly displaced toward the tympanic cavity, and *a*, connective tissue and capillary between the dura of the middle cranial fossa and the mucosa of an infected pneumatic cell.

toward the middle ear. The antrum was narrowed by edematous mucosa; the horizontal semicircular canal was normal. The pneumatic cells of the remnant of the mastoid process were filled with pus. There was osteitis of the superior and posterior wall of the bony external canal.

The ductus and saccus endolymphaticus formed a tube, the axis of which ran almost parallel to the axis of the crus commune. Medialward the duct turned toward the posterior cranial fossa so that the axis formed an angle of less than 90 degrees with the axis of the crus commune. At the curve[5] the endolymphatic duct, as well as the vestibular aqueduct, formed an ampulla-like dilatation (figs. 2 and 4), which became rapidly narrower toward the saccule and which abruptly changed into a slitlike lumen toward the cranial opening of the aqueduct. In the area of the dilatation, the structure of the endolymphatic duct was similar to the structure of a part of the endolymphatic duct in guinea pigs; the latter structure Guild[6] called pars intermedia, although in the presented case the epithelium in this area was cubical, not columnar, as it is in guinea pigs (fig. 4).

Close to the saccule the wall of the endolymphatic duct consisted of firm connective tissue, which served as a periosteum. Toward the lumen the connective tissue became loose and edematous and was covered by low cubical epithelium, of the same kind as Siebenmann[7] noted in the normal endolymphatic duct in man. In the comparatively large lumen were erythrocytes and lymphocytes. In the ampulla-like dilatation there was, again, the periosteum, which originated from the dura of the posterior cranial fossa (fig. 4 *c*). Within the periosteum was a small number of dilated capillaries, presenting a perivascular infiltration. In addition, there were masses of yellow and brown pigment, apparently remnants of hemorrhages, although not verified by a test for iron. The osseous walls of the vestibular aqueduct occasionally showed lacunas, indicating a bone absorption of low intensity. The rest of the osseous wall was separated from the periosteum by a fine blue *Grenzscheide*. In this area the walls of the endolymphatic duct formed numerous villi and polypoid excrescences, which bulged into the lumen of the duct (fig. 4). Frequently, two villi facing one another fused, after having lost their epithelium. This caused the lumen of the duct to be divided into several small cavities, which were either empty or filled with serous exudate. The epithelium was low and cubical.

The villi consisted of loose connective tissue, which contained a glasslike edema fluid, or of areolar tissue, which was crowded with specific cells (fig. 4). The cells were of various sizes, were round or oval and showed much cytoplasm, crowded with yellow granules. The nucleus was comparatively small, contained a

5. The osseous channel of the vestibular aqueduct starts at the posterior surface of the petrous bone and runs within the petrous bone in an oblique direction, mesialward and upward toward the area where the anterior and the posterior semicircular canal fuse and form the crus commune. In this area the channel forms a curve behind the crus commune and runs in an oblique direction downward toward the junction of the crus commune with the osseous vestibulum. The osseous channel harbors the membranous endolymphatic duct, which takes the same course as the osseous channel.

6. Guild, S. R.: Observations upon the Structure and Normal Contents of the Ductus and Saccus Endolymphaticus in the Guinea-Pig (Cavia Cobaya), Am. J. Anat. **39**:1, 1927.

7. Siebenmann, F.: Anatomische Untersuchungen über den Saccus und Ductus endolymphaticus beim Menschen, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **13**:59, 1919.

considerable amount of chromatin and was either centrally or eccentrically located. These cells were undoubtedly phagocytes. They were loosely scattered between the connective tissue fibers (fig. 5), which were torn by the edema, or they were piled up and may have produced bulging of the epithelium toward the lumen, or they showed an epithelium-like arrangment. They did not accumulate around the dilated blood vessels, and they almost disappeared where there was perivascular infiltration of lymphocytes or recent hemorrhage. They were not to be found in the lumen of the duct.

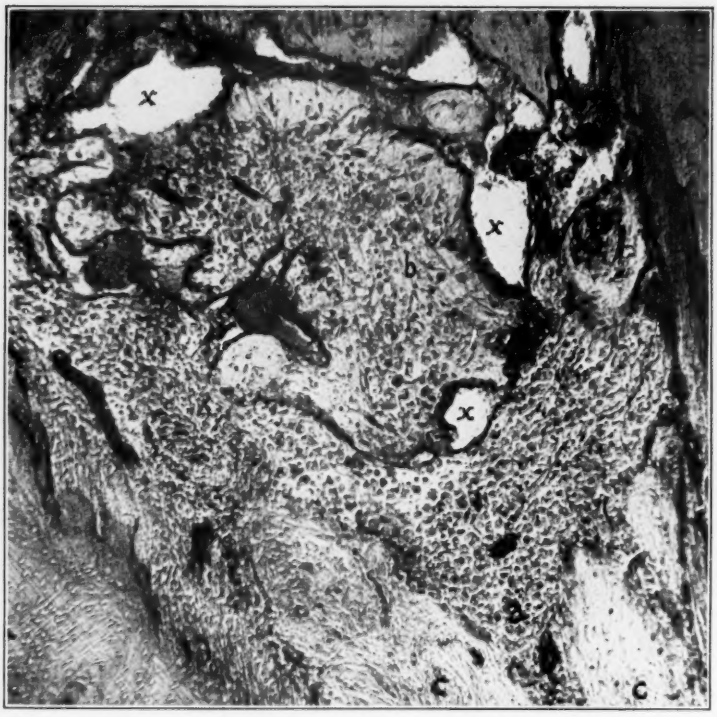

Fig. 4.—Ductus endolymphaticus of figure 2 in higher magnification. The lumen of the duct is obliterated, to a great extent, by pronounced edema of the walls (*b*). Only remnants of the lumen (*x*), partially filled with serous exudate, are noticeable. The edematous walls of the duct are filled with gitter cells, which are presented in figure 5 in higher magnification. The gitter cells seem to accumulate toward the funnel-like narrowing of the duct (*a*), where they enter the narrow cerebral portion of the duct. Firm connective tissue (*c*) surrounds the cerebral portion of the duct.

Toward the dura, the vestibular aqueduct became narrow, and the endolymphatic duct presented a slitlike lumen. A great number of the phagocytes entered this part of the duct, and, because there was not much space available, the cells were

firmly pressed against each other (fig. 4). Farther cranialward the wall of the duct consisted of firm connective tissue of dural origin. In this part there were only a few phagocytes and several corpora arenacea. The lumen and the walls of the endolymphatic sac proper did not contain any pathologic material.

Right Temporal Bone: Here, likewise, was a severe infection of the apex of the pyramid and of the perilabyrinthine cells (fig. 1). The area of destruction had essentially the same localization with respect to the internal ear as it had on the left side, but apparently this area was not as extensive as it was on the left. Furthermore, the area consisted almost exclusively of firm connective tissue and

Fig. 5.—Gitter cells within the membranous wall of the endolymphatic duct; *a* indicates epithelium, *x*, exudate within the lumen of the duct.

newly formed bone. There were only a few small abscesses within the connective tissue. In the cochlea there were essentially the same changes as on the left side. In the tube there was pus and the mucosa was considerably swollen. The epithelium of the mucosa was normal.

Comment.—The following pathologic changes were seen on both sides: (*a*) acute infection of the mucosa of the tube and tympanic cavity; (*b*) extensive osteitis of the apex of the pyramid and of the periosteal layer of the capsule of the internal ear, which was more pronounced on the left side than on the right, with large abscesses on the left; (*c*) acute purulent infection of the perilabyrinthine cells, and (*d*) acute serous labyrinthitis.

In addition, there were the following conditions on the left side: (*a*) dislocation of the stapes toward the tympanic cavity; (*b*) acute infection of the cells of the mastoid process; (*c*) osteitis of the superior and posterior wall of the external auditory canal; (*d*) inflammatory edema of the wall of the endolymphatic duct, and (*e*) accumulation of phagocytes in the wall of the endolymphatic duct.

GENERAL COMMENT

In the presented case, bilateral acute otitis media developed during the first week of scarlatina. Two days after the first appearance of the aural symptoms, the child became deaf on both sides. Then, necrosis developed in the pharynx; the temperature rose, and there was pronounced leukocytosis, with moderate shifting to the left. Ten days after the appearance of the first aural symptoms the child became stone deaf on the left side and on the right could hear only noises. There was no spontaneous nystagmus, and the caloric test revealed hypoexcitability on both sides. Antrotomy was performed on both sides. On the right, the infection of the mastoid was cleared in due time, but the discharge from the left mastoid did not cease. Seven days after the antrotomy there were paralysis of the left abducens nerve and anesthesia of the left trigeminal nerve, which, however, were not associated with pain. This was followed by slight paresis of the left facial nerve and paresis of the soft palate. In the meantime, thrush of the mucosa of the lips and the cheeks, extreme swelling of the glands and symptoms of nasal diphtheria were noted. Finally, fatal bronchopneumonia developed about five weeks after the operation. Autopsy revealed diphtheria of the myocardium and bonchopneumonia. There were edema and hyperemia of the brain, but there was no meningitis.

In this case severe scarlatinous infection was associated with nasal diphtheria. In such instances the infection usually travels along the eustachian tube into the tympanic cavity, causing necrotic otitis media. In the presented case, however, not necrotic, but common, otitis media developed in a patient with scarlatina and diphtheria. The otitis media was the source of several complications. There was bilateral mastoiditis. From the antrum and the epitympanum, the infection traveled (*a*) into the pneumatic cells of the pyramid, causing petrositis, and (*b*) into the inner ear, causing serous labyrinthitis.

Since both temporal bones were very well pneumatized, the petrositis extended actually to the petrous apex on both sides. The general condition of the child was poor; yet there was spontaneous clearing of the petrositis on the right side (fig. 1) almost in its entire extension, owing to the formation of connective tissue and bone. On this side the petrositis did not extend beyond the boundaries of the petrous bone and there were no definite clinical symptoms of petrositis, although

the infection extended over a large area of the pyramid. Antibiotics and sulfonamide drugs were not available at this time. On the left side the petrositis extended over a larger area than on the right. On the left, the petrositis did cause clinical symptoms referable to the abducens, trigeminal, glossopharyngeal and vagus nerves. Among the symptoms of trigeminal involvement pain was absent, but there was anesthesia of the skin of the cheeks and of the cornea, the corneal insensitivity producing ulceration of the cornea. The importance of such anesthesia in the absence of trigeminal pain was stressed by Grabscheid.[8] On the left side the petrositis was likewise cleared, but only to a certain extent (fig. 2). There were several abscesses of various sizes in the scar tissue close to the dura. Nevertheless, the infection did not enter the dura on that side and no meningitis was observed at autopsy. This observation indicates that purulent petrositis has a marked tendency toward spontaneous clearing, particularly in children, even in the presence of a poor general condition. In these instances operation on the petrous apex is usually not necessary; it may even be harmful. This point of view I have stressed previously.[9]

The next complication of the acute otitis was serous labyrinthitis characterized by an accumulation of a serofibrinous exudate in the endolymphatic and perilymphatic spaces (figs. 1, 2 and 3). There apparently had been a greater amount of fluid in the inner ear during life, because there were dislocation of the stapes and bulging of the membrane of the round window toward the tympanic cavity, signs indicating an increased pressure within the inner ear. The specimen was not well enough preserved to permit one to draw conclusions concerning Corti's organ; however, there was no pronounced diminution of the ganglion cells and nerve fibers in the cochlea on either side except in the basilar portion, where a moderate amount of ganglion cells had been destroyed. The maculas and cristae on the left side were normal; there was only a very small loss of nerve fibers in the left utricular nerve. The maculas and cristae on the right side were not examined.

There was inflammatory edema of the wall of the left endolymphatic duct, which extended from the opening in the saccule to the curve of the duct and disappeared toward the cranial opening of the vestibular duct (fig. 4). The inflammation of the wall of the duct had caused mild absorption of the osseous channel. When the lining epithelium was absent, the polypoid excrescences, caused by the edema of the wall, fused, with resulting partial or complete obliteration of the lumen.

8. Grabscheid, E.: Un contributo alla clinica delle malatti inflammatorie della punta della rocca, Rassegna ital. di oto-rino-laring. **5**:148, 1931.

9. Brunner, H.: Intracranial Complications of Ear, Nose and Throat Infections, Chicago, The Year Book Publishers, Inc., 1946.

Of particular interest was the presence of a great amount of phagocytes in the areolar tissue of the wall of the duct (figs. 4 and 5). The cytoplasm of the cells was filled with granular yellow material. Although no specific staining had been performed, it is likely that the cells contained lipids. Cells of this type are known to arise from the reticuloendothelial system, present in all types of tissue. In the brain, these cells are produced by the microglia (mesodermal) and the perivascular histiocytes and are called gitter cells, or *Körnchenzellen*. In the ear, these cells were not particularly studied, but they are commonly seen in the exudate of acute otitis media and of serous labyrinthitis.

In the lumen of the endolymphatic duct of guinea pigs, Guild [6] noted numerous cells, among them phagocytes, which morphologically were similar to the phagocytes in the presented case. Siebenmann [7] emphasized that in the endolymphatic duct in man there are no abnormal cells in the lumen or in the subepithelial tissue. For this reason the phagocytes in the presented case must be considered pathologic, and the conclusion must be drawn that the phagocytes accomplish the resorption of the serous exudate in the internal ear. In favor of this concept are the following facts: 1. In the acute stage of serous labyrinthitis, phagocytes do appear in the internal ear. 2. In the presented case there had been acute serous labyrinthitis, but at the time of death the exudate had disappeared to a great extent from the inner ear. Thus, the case proves that in serous labyrinthitis the exudate is carried away from the internal ear by phagocytes.[10] It is obvious that the phagocytes which carry the exudate arise in the internal ear from the reticuloendothelial system, as they do in the rest of the body. This is proved by the findings in Gaucher's disease, in which these phagocytes arise from the histiocytes in the marrow spaces of the temporal bone (Brunner [11]).

The presence of phagocytes in the subepithelial tissue at the curve of the endolymphatic duct, on the one hand, and the absence of cells and of a massive exudate in the inner ear, on the other, indicate that there must be a flow of the phagocytes, or of the exudate or of both from the internal ear toward the endolymphatic sac. This flow was temporarily stopped at the curve of the duct because there was a dilatation which was continued toward the cranial fossa by an isthmus-like narrowing of the endolymphatic duct. This concept explains the

10. It is likely that these phagocytes are very frail elements, like the gitter cells in the brain. They make their appearance when the serous labyrinthitis is going to subside, and they probably disappear rapidly when the inflammation has subsided.

11. Brunner, H.: Ueber das Vorkommen von Gaucherzellen im Felsenbeine, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:60, 1928.

absence of phagocytes in the part of the endolymphatic duct which extended from the vestibule to the curve of the duct, although there was considerable edema of the wall in this part of the duct.

From these findings the following conclusions must be drawn: In serous labyrinthitis there is a flow from the internal ear toward the endolymphatic sac. This flow carries the inflammatory products away from the inner ear. It is a question of secondary importance whether the inflammatory products are resorbed by phagocytes within the inner ear and the phagocytes are then carried into the endolymphatic duct, or whether the exudate flows into the duct and is resorbed by phagocytes which arise in the wall of the duct. The latter concept seems more likely. In either case, it is certain that in serous labyrinthitis a resorption takes place in the endolymphatic duct. This concept is in agreement with the view of many investigators, particularly that of Guild,[12] concerning the function of the normal endolymphatic duct, although it must be kept in mind that the observations made on pathologic cases do not necessarily permit conclusions concerning physiologic conditions.

Serous labyrinthitis is not infrequent in scarlatina otitis. Nager,[13] Mayer,[14] Manasse,[2] Zange[15] and others have reported cases of this type. Nager was the first to show that in these instances there is no fistula in the window or in other parts of the lateral wall of the labyrinth; the infection travels, rather, from the tympanic cavity through the inflamed but not perforated membranes of the windows into the internal ear. According to the terminology of Ruttin,[3] this type of labyrinthitis is, therefore, called diffuse serous induced labyrinthitis.

The presented case differs in several aspects from the reported cases. In this case there was severe petrositis, which eventually may be considered the source of the serous labyrinthitis. This concept, however, is not correct, at least not for the left side, which was thoroughly examined, for the petrositis had involved almost exclusively the periosteal layer of the bony capsule, and thus the entire endochondral layer intervened between the area of the petrositis and the internal ear. Moreover, in the other cases the serous labyrinthitis was caused almost exclusively by the necrotic type of otitis complicating scarlatina. In this case there was no necrotic otitis media, despite the associated nasal diphtheria. On the contrary, the tympanic mucosa was extremely

12. Guild, S. R.: The Circulation of the Endolymph, Am. J. Anat. **39**:57, 1927.

13. Nager, F.: Eine statistische Studie über die scarlatinöse Erkrankung des Gehörorgans, Wiesbaden, J. F. Bergmann, 1908.

14. Mayer, O.: Zur Entstehung der sogenannten Labyrinthitis serosa im Verlaufe akuter Mittelohrentzündungen, Monatsschr. f. Ohrenh. **43**:1909, 1909.

15. Zange, J.: Pathologische Anatomie und Physiologie der mittelohrentspringenden Labyrinthentzündungen, in Körner, O.: Die Ohrenheilkunde der Gegenwart und ihre Grenzgebiete, Wiesbaden, J. F. Bergman, 1919, vol. 10.

edematous (fig. 3), and there was no defect of the epithelium except in those areas where operation was performed. The epithelium was higher than is usual with normal conditions, a common finding in acute otitis media. These observations prove that in scarlatina serous labyrinthitis may occur without being caused by necrotic otitis media.

Ultimately, in most of the reported cases the annular ligament and the membrane of the round window were at least partially inflamed or necrotic, and there was severe inflammation in the area of the window. This indicates that in the reported cases there was perhaps a secondary diffuse serous labyrinthitis, in contradistinction to this case, in which, on the left side, induced serous labyrinthitis was discovered. The question is whether the annular ligament and the membrane of the round window, which at autopsy were found to be normal (fig. 3), had ever been inflamed during the long course of the illness. This cannot be answered; however, if they had been inflamed, evidence had completely disappeared at the time of autopsy.

The symptoms of induced diffuse serous labyrinthitis consist of dizziness and nystagmus to the opposite side, at least of second degree intensity. There is no symptom of fistula. Hearing and caloric and turning reactions are not entirely absent, or, if they are, they return completely or partially after the labyrinthitis is cleared. In the presented case the symptoms were different. There was, immediately following the paracentesis, bilateral diminution of hearing, which within four days increased to complete deafness on both sides. Labyrinthine symptoms—dizziness and spontaneous nystagmus—were absent, and the caloric reaction was diminished on both sides but was not absent. This elicitation of the caloric reaction suggested the diagnosis of serous labyrinthitis, because in purulent labyrinthitis the caloric reaction cannot be elicited. This diagnosis was confirmed by the microscopic examination. For this reason the presented case proves the following points:

1. Acute deafness in scarlatina may be caused by bilateral otitis media plus bilateral diffuse serous labyrinthitis.

2. The otitis media which causes the serous labyrinthitis may be common acute otitis; it need not be necrotic otitis.

3. The serous labyrinthitis which causes the deafness may be an induced labyrinthitis, i. e., labyrinthitis caused by the passage of toxins through the intact windows.

4. In cases of induced serous labyrinthitis, the cochlear nerve is normal (on section, with the hematoxylin-eosin and Van Gieson stains), and the spiral ganglion is almost normal.

5. An induced serous labyrinthitis may cause deafness but may fail to produce spontaneous labyrinthine symptoms.

The fact that serous labyrinthitis eventually causes complete deafness was emphasized by Herzog,[16] who asserted that even inflammatory changes of the endolymph not associated with destruction of the sensorial epithelium may cause complete and acute deafness. Herzog's observation was confirmed by Ruttin, Neumann and others, but it was added that in these instances the acute deafness is associated with acute labyrinthine symptoms and that the complete deafness is usually transitory. In the presented case there were no acute labyrinthine symptoms. This is unusual, but it is conceivable. The inflamed endolymph in serous labyrinthitis exerts various kinds of influence on the sensorial cells: (*a*) a mechanical influence, due to pressure on the sensorial cells; (*b*) a toxic influence, due to toxins within the endolymph, and, perhaps, (*c*) a chemical influence, due to increased content of proteins in the endolymph. It cannot be predicted to which degree these factors will damage the sensorial organs in an individual case, or which sensorial organs they will injure more and which they will injure less. This may be the reason for the variability of clinical symptoms in serous labyrinthitis.

The question whether hearing in cases of serous labyrinthitis must necessarily be restored is related to the concept of serous labyrinthitis in general. There is by no means unanimity on this concept. Some otologists consider serous labyrinthitis a morbid entity; others regard it simply as the prodromal stage of a purulent infection. Personally, in agreement with a great many otologists, I have always considered serous labyrinthitis (like serous meningitis) a morbid entity which, even after prolonged duration, does not necessarily change into purulent labyrinthitis. This takes well into consideration the fact that purulent labyrinthitis eventually may set in, producing a serous exudate. However, in such instances the patient is severely ill, the meninges are very soon involved and the change from serous to purulent exudate is rapid. The presented case proves the accuracy of this view. The child lived for more than five weeks after the onset of deafness. Nevertheless, the labyrinthitis was serous and not purulent, although there had been enough time for a change to occur from serous to purulent exudate and although no sulfonamide drugs or antibiotics were given.

The concept of serous labyrinthitis as a morbid entity does not imply that there must invariably be a restoration of hearing. I believe, rather, that the serous labyrinthitis per se may cause not only transitory but permanent deafness, although partial, and occasionally complete, restoration of hearing is more frequent. In this case hearing was not restored during the five and one-half weeks of illness before the child died. The question is whether hearing would have been restored had

16. Herzog, M.: Labyrintheiterung und Gehoer, Munich, J. F. Lehmann, 1907.

the child lived. The answer is purely speculative. The facts that within a period of more than five weeks only moderate atrophy of the ganglion of the basilar portion had developed and that during this period nothing occurred to indicate an organization of the exudate render it likely that hearing would have been restored, though probably not to a normal extent. In fact, this is what frequently happens in cases of survival.

CONCLUSIONS

1. In serous labyrinthitis the exudate is carried away from the internal ear—at least to a great extent—by phagocytes.

2. In serous labyrinthitis resorption of inflammatory products takes place in the endolymphatic duct.

3. In scarlet fever serous labyrinthitis may occur without being caused by necrotic otitis media.

4. Acute deafness in scarlatina may be caused by bilateral otitis media plus bilateral diffuse serous labyrinthitis.

5. The serous labyrinthitis may be an induced labyrinthitis, i. e., labyrinthitis caused by the passage of toxins through the intact windows.

6. An induced serous labyrinthitis may cause deafness but may fail to produce spontaneous labyrinthine symptoms.

7. Serous labyrinthitis per se may cause not only transitory deafness but also permanent diminution of hearing.

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UNILATERAL PARALYSIS OF THE LARYNX

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THIS article is an attempt to correlate the material published since 1913 on the subject of unilateral paralysis of the larynx. The observation of 3 cases of unilateral laryngeal paralysis within one month, in all 3 of which the left cord was involved and the etiologic factors were different, suggested a survey of the literature on this subject, which, on investigation, proved to be voluminous but widely scattered. The following paragraphs attempt to bring together the pertinent facts discovered.

There are five types of laryngeal paralysis: (1) congenital, (2) central, (3) peripheral, (4) myopathic and (5) psychogenic. The congenital type which is present at birth, is rare and is bilateral. The central type, which is comparatively rare, is divided into the cortical and bulbar subgroups. Paralysis of cortical type is very rare, and, since the center for the larynx in the cortex is bilateral, a bilateral lesion in this area would be necessary to produce a paralysis of the larynx and the paralysis would be bilateral. The second form of central paralysis is that due to pathologic changes of the bulbar region and the jugular foramen. The peripheral type may be produced by any lesion affecting the vagus nerve down to and including the recurrent nerve; the myopathic and psychogenic types are self explanatory. It is with the bulbar and peripheral types that this paper is concerned.

Unilateral paralysis of the recurrent laryngeal nerve is not uncommon; unilateral paralysis of the larynx due to involvement of the medulla or jugular foramen is rare. Hall [1] in 163 cases found that only 10 were due to a lesion of the medulla or associated cranial nerves. Thomson, cited by New,[2] found that in a series of 360 cases there was only 1 attributable to this cause, and this case was due to an extracranial growth involving the vagus close to its exit from the foramen.

Read before the Philadelphia Laryngological Society, April 4, 1949.

1. Hall, H. D.; Ferrier, D., and Permawan, W., in discussion on the Etiology of Unilateral Paralysis of the Recurrent Laryngeal Nerve, Proc. Roy. Soc. Med. **6**:139, 1913.

2. New, G. B., and Childrey, J. H.: Paralysis of Vocal Cords: Study of Two Hundred and Seventeen Medical Cases, Arch. Otolaryng. **16**:143 (Aug.) 1932.

Guder and Dufour,[3] in a series of 63 cases, found none that could be called bulbar in nature.

The etiologic factors involved in the production of laryngeal paralysis may be grouped into four major divisions: (1) the nervous system; (2) diseases of the respiratory tract; (3) circulatory diseases; (4) diseases of the adjacent areas (Lederer).[4] This classification is much like that of Hall,[1] who listed ten neurogenic causes and fifteen that are not neurogenic but cause pathologic conditions of the recurrent nerves. Lederer listed sixteen causes due to primary disease of the nervous system.

These may be considered in more detail. The nervous system affords numerous variations as, for instance, trauma due to accidents and injuries with dangerous weapons, the effects of which may be direct or indirect; neuritis, both exogenous and endogenous—the former caused by hair dyes, drugs, etc., and the latter by scarlet fever, influenza, malaria, typhoid, syphilis and the like. General nervous system disorders include progressive bulbar palsy, tumor of the brain, multiple sclerosis, syringomyelia, tabes, dementia paralytica, central nervous system syphilis, bulbar poliomyelitis, hemorrhage of the brain and the bulb with softening of nervous tissue, and sympathetic nervous system dysfunction.

The diseases of the respiratory tract include tumors of the lung and tuberculosis of the apex of the lung causing a thickened pleura, enlarged lymph nodes of the bronchial tree and mediastinal tumors.

The circulatory conditions most frequently observed are enlarged heart causing a dilated ventricle or left auricle to press on one of the recurrent nerves, aneurysm of the aorta, pericarditis, mitral stenosis and pericardial adhesions.

Diseases of the adjacent areas consist of tumors of the neck—retrosternal and intrathoracic thyroid, and malignant and benign tumors of the thyroid gland—and Hodgkin's disease. While thyroid surgery cannot be classed as a disease, injury of the recurrent nerves during an operation on the gland is one of the main factors in paralysis of the larynx.

Clerf [5] has stated "Paralysis of vocal cord is rarely caused by disease of the larynx. The causes must be found within the thoracic cage and mediastinum, neck or cranial cavity." Even after the most careful study, a great many of these cases remain undiagnosed as far as the etiologic agent is concerned.

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3. Guder and Dufour, cited by Permawan, in discussion on the Etiology of Unilateral Paralysis of the Recurrent Laryngeal Nerve, Proc. Roy. Soc. Med. **6**:152, 1913.

4. Lederer, F.: Diseases of the Ear, Nose and Throat: Principles and Practice of Otorhinolaryngology, ed. 5, Philadelphia, F. A. Davis Company, 1946, p. 751.

5. Clerf, L. H.: Paralysis of the Larynx, J. Michigan M. Soc. **46**:441 (April) 1947.

The left recurrent nerve is the one most frequently involved in unilateral paralysis in the ratio of about 4 to 1 because of its peculiar anatomy. Hall [6] stated that "if malignant disease of the esophagus be excluded, it may be baldly stated that the right recurrent nerve is hardly ever paralysed as a result of pressure in the mediastinum."

In summary of the etiologic agents causing this condition, it is probable that hemorrhage of the bulb is the commonest cause of the bulbar type of lesion; carcinoma of the upper third of the esophagus the cause of a lesion in the hypopharynx and aneurysm of the aorta the commonest cause of a lesion in the lower part of the neck, with surgical injury to the recurrent nerve in the neck following closely.

With the foregoing etiologic factors in mind, it is evident that any one or all three of the functions of the larynx will be affected in paralysis of the organ—respiration, phonation and deglutition.

According to New,[7] in unilateral paralysis due to injury to the recurrent nerve the affected cord is in the median or paramedian position. The cord is bowed, relaxed and lower than its fellow. If the paralysis is due to injury of the recurrent nerve only, the paralyzed cord is found in the median position, but if it is due to injury of the vagus nerve the cord (injury at the jugular foramen) is found in the cadaveric position. The symptoms vary with the type of paralysis and the extent. In unilateral lesions the voice is seldom impaired, the only alteration being a quality of coarseness which becomes more pronounced after prolonged use. There is usually no dyspnea unless the patient exerts himself more than normal, there being enough opening of the glottic chink for normal respiration. According to Semon's Law, the abductor muscles are first affected and the cord lies in the median position. Then comes a degeneration of the adductor muscles, and the cord recedes to the paramedian position when both sets of muscles are incapacitated. The normal cord attempts to cross the midline on phonation. This is considered an attempt of the physiologic sensibility of the larynx to compensate for the injured cord.

The prognosis of paralysis of the larynx is variable. Mulligan [8] has stated that in patients with unilateral paralysis following thyroidectomy the voice definitely improved in six weeks but after prolonged use of the voice only a whisper could be elicited. Complete recovery was seen in only 1 case. New and Childrey [9] stated that a cord fixed medianly may recover completely but will remain fixed medianly and a

6. Hall, Ferrier and Permawan,[1] p. 142.

7. New, G. B., and Childrey, J. H.: Paralysis of Vocal Cords: Study of Two Hundred and Seventeen Medical Cases, Arch. Otolaryng. **16**:145 (Aug.) 1932.

8. Mulligan, E. J.: Prognosis of Laryngeal Paralysis Following Thyroidectomy, Arch. Otolaryng. **35**:732 (May) 1942.

9. New and Childrey,[7] p. 155.

cord in the cadaveric position may recover but in a few months will swing to the median position. New found that position of the cords had some relation to the time of recovery. With a cord in the midline the recovery time averaged seventeen months, whereas with the cord in the cadaveric position the recovery time was only ten and one-half months. Smith, Lambert and Wallace, writing in the *Edinburgh Medical Journal*,[10] took issue with New and Childrey on the position of the cord. In the majority of their cases the cord was in the cadaveric position, and they felt that it is difficult to conceive that a concurrent superior laryngeal paralysis is always present to account for this, as New and Childrey have postulated. They also stated that the commonest causes of paralysis of the recurrent nerve in their cases were aneurysm of the aorta and mediastinal tumors, and they felt that Childrey and New were too optimistic regarding the ultimate outcome in these cases. Suehs [11] analyzed the records of 270 cases of laryngeal paralysis over a period of eighteen years and concluded that carcinoma was the commonest cause of the unilateral type, that surgical procedures in the thyroid gland were the most frequent cause of the bilateral type and that, whatever the cause, the prospect of recovery is poor at best but increases somewhat if the etiologic agent can be discovered.

Imperatori and Burger have thoroughly covered the subject of the associated paralyses of the larynx. Imperatori [12] mentioned three variations of the Avellis syndrome. He stated that the original syndrome of Avellis consisted of the following conditions: (1) paralysis of the vocal cord and soft palate and partial paralysis of the constrictor muscles of the pharynx and esophagus; (2) loss of pain and temperature sense of the contralateral side of the body below the interauricular line with retention of all other somatic sensibilities, and (3) endarteritis. In describing the variations of the syndrome, he mentioned that of Schmidt, in which to the already described dysfunctions there is added a lower motor neuron paralysis of the sternomastoid and trapezius muscles. In addition to these findings of Avellis and Schmidt, if paralysis and atrophy of the ipsilateral side of the tongue are added, the result is Jackson's syndrome. If lateropulsion and loss of sensibility of the homolateral side of the face are added, Tapia's syndrome is present.

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10. Smith, A. B.; Lambert, V. F., and Wallace, H. L.: Paralysis of the Recurrent Nerve: Survey of Two Hundred and Thirty-Five Cases, Edinburgh M. J. **40**:344 (July) 1933.

11. Suehs, O. W.: Paralysis of the Larynx: Study of Two Hundred and Seventy Cases, Texas State J. Med. **38**:665 (March) 1943.

12. Imperatori, C. J.: Syndrome of Avellis: Report of Three Cases, Arch. Otolaryng. **1**:277 (March) 1925.

Burger,[13] in the Semon Lecture of 1934, thoroughly discussed the whole subject of the associated paralyses of the vocal cord and suggested that a classification into four syndromes on an anatomic basis would be helpful in understanding the various findings. This is much like that cited in Lederer's textbook.

The existence of associated paralyses of the vocal cord is of great importance for localization of lesions, but it is seldom that more than a rough distinction between a high and a low injury of the vagus and a distinction between an injury of the recurrent and of the vagus nerve are discerned. In pure recurrent nerve paralysis the cord is the only structure affected; in lesions of the vagus nerve one may find in addition to paralysis of the cord, hemianesthesia of the larynx, hemiparalysis of the palate, acceleration of the pulse, abolition of the oculocardiac reflex and associated paralyses already described.

Burger's Classification of Associated Paralyses

	Syndrome	Nerves Involved	Vagus Involvement
I.	Bulbar nerve syndrome	Fifth to twelfth cranial	High
II.	Jugular foramen syndrome	Ninth to twelfth cranial; sympathetic	High
III.	Parapharyngeal space syndrome	Ninth, tenth, seventh, twelfth cranial; sympathetic	Low
IV.	Vocal cord-diaphragm syndrome	Tenth cranial; phrenic; sympathetic	Low

It was thought originally that the various paralyses of the associated type were due to hemorrhage into the nucleus ambiguus in the medulla and into surrounding nuclei, but it was thought impossible that such a thing could happen without affecting other structures in the neighborhood, particularly when one takes into consideration the large extent of the ninth, tenth, eleventh and twelfth nerve nuclei.

No other class of paralysis of the bulbar nerves is so typical as that of the ninth, tenth and eleventh. In searching for a cause of this syndrome, the lateral sinus and its thrombosis come to mind as the first possibility; yet the literature on sinus thrombosis has never mentioned the three complete, according to Burger.[14]

It is evident that it would be very easy to have trouble in the region of the jugular bulb. Pachymeningitis of the sinus, involving the bulb; too tight packing of a mastoid wound after an operation on the lateral sinus; tumors of the parapharyngeal space, and lesions of the external and middle ear—all have their place in the causation of this entity.

13. Burger, H.: Associated Paralyses of Vocal Cord (Semon Lecture, 1933), J. Laryng. & Otol. **49**:1 (Jan.) 1934.

14. Burger,[13] p. 15.

REPORT OF CASES

The patients whose cases are briefly reported were all seen in the outpatient laryngeal clinic of the Philadelphia General Hospital.

CASE 1.—A man complained of hoarseness of two years' duration and inability to use his left shoulder properly. On examination there was a complete paralysis of the left cord with slight bowing, and the cord itself was slightly off the median line. The soft palate was not paralyzed but appeared to be lacking in sensitivity. The left trapezius muscle showed atrophy, and there was some limitation of movement. There was a mass in the left supraclavicular space 1 inch (2.5 cm.) above the clavicle. The patient was referred to the radiology department for roentgenograms of the skull, chest and cervical and thoracic portions of the spine and from there was sent to the neurologic department for examination. The following report was submitted by the neurologist:

"The cranial nerves are normal except for a questionable miosis of the left eye (anisocoria) and a questionable right central seventh. There is wasting of the left trapezius, but only a little loss of motor power. The sternomastoid on the left is not involved. Sensory and cerebellar functions are normal. There is no weakness of the extremities; deep tendon reflexes are normal; there is a slight wasting of the left triceps muscle with no functional loss. There seems to be a round firm mass in the left supraclavicular fossa. The paralysis of the left cord plus the atrophy of the trapezius and tricep muscles can be explained by a local mass lesion. The questionable Horner syndrome on the left gives rise to a question of a sulcus neoplasm. Lack of involvement of the sternomastoid muscle and absence of long tract signs make a central lesion unlikely."

CASE 2.—A man, 76 years of age, complained of hoarseness and dyspnea on exertion, with reduction of the voice to a whisper when he talked for any length of time. Examination revealed paralysis of the recurrent laryngeal nerve on the left side, with no evidence of any other local lesion. He stated that his trouble had started two weeks prior to coming to the hospital. A roentgenogram of the chest showed a large saccular aneurysm involving the distal horizontal and upper descending portions of the aorta. There were atheromatous plaques in the sac. The trachea was displaced to the right and the left side of the diaphragm elevated.

CASE 3.—A 26 year old woman sustained an injury to the neck in an automobile accident on Feb. 29, 1948. She was admitted to the hospital, and it was noted at the time of admission that she had had an injury to the larynx with swelling of the left arytenoid muscle and vocal cord and dysphagia. A roentgenogram of the neck at that time did not reveal the nature of the injury to the larynx. There was considerable edema of the whole larynx, especially the left side. On March 11 the right side of the larynx was beginning to move better, but the left side was very sluggish and the ventricular band bulged into the interior of the larynx. The edema was clearing. She was dismissed from the hospital on March 12 with swallowing nearly normal, laryngeal edema decreased and hoarseness still present.

When she was seen again, one week later, the voice was still a whisper. The left cord was completely paralyzed and just off the midline. There was still some swelling of the ventricular bands, but the left band had receded from the interior of the larynx. There was a definite crepitus felt along the area of the junction of the thyroid and cricoid cartilages, and this was the probable site of injury of the left recurrent nerve causing the paralysis. Roentgenologic evidence of laryngeal injury was unobtainable.

SUMMARY AND CONCLUSIONS

Some of the literature dealing with paralysis of the larynx has been reviewed, and 3 typical cases of unilateral laryngeal paralysis are reported.

Unilateral paralysis is largely a medical problem. Exact diagnosis of the cause of paralysis is not made in 20 per cent of the cases. The therapy of this condition is unsatisfactory; it should be directed toward correction of the lesion causing the paralysis if it can be found. The prognosis is poor at best but is improved somewhat if the etiologic agent is known and proper treatment can be instituted.

Union Trust Building.

IDIOPATHIC PARALYSIS OF THE VOCAL CORD

Fixation of the Vocal Cord Without Anatomic Lesion

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UNILATERAL paralysis of the vocal cord producing hoarseness and occasionally narrowing of the glottis is frequently seen by the laryngologist. In most patients with this condition the inferior laryngeal branch of the vagus nerve has been injured by an organic lesion. The left inferior laryngeal (recurrent laryngeal) nerve is more frequently affected than the right because of its greater anatomic complications. Thus, as it courses about the arch of the aorta, the recurrent laryngeal nerve becomes susceptible to pressure from an aortic aneurysm in varying degrees. Consequently, a harsh, strident or hoarse voice as an early symptom of such a lesion brings the patient to the attention of the laryngologist, who readily recognizes the mediastinal significance of the paretic left cord. One may then complete the diagnosis by looking for a tracheal tug, abnormal surface pulsation near the sternum and roentgenographic and fluoroscopic evidence of increased aortic width.

Other causes of paralysis of the vocal cord are well known. Pressure exerted by enlargement of the thyroid gland, diseased lymph nodes, mediastinal growths, pleurisy, tumors of the esophagus, mitral stenosis or apical infiltration is not infrequently the cause of paresis of a vocal cord. Surgical injury, as in thyroidectomy, has too often resulted in permanent damage to the inferior laryngeal nerves. In some cases a form of toxic neuritis, due to various toxic agents, such as lead, alcohol, arsenic or atropine, has been observed. However, I wish to emphasize here an important differential diagnosis: that of arthritis or perichondritis of the thyroarytenoid articulation, which not infrequently goes unnoticed during indirect laryngoscopy and which results in a wasteful search for a deeper pathologic condition to explain the partial or complete immobilization of the cord.

The subject of this paper is the idiopathic paralysis of a vocal cord. This form of paralysis is called idiopathic when the nerve per se is involved, uninfluenced by pressure from without. It is assumed that the involvement is a neuritis which affects all the fibers of the inferior laryngeal nerve, immobilizing the vocal cord in the medial position

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between maximum abduction and adduction, the so-called cadaveric position. I saw 5 such cases with complete immobilization of the vocal cord, and, by coincidence, all on the right side.

The clinical records of the patients were surprisingly alike. The patient quite suddenly noted a change in the quality of the voice, accompanied with abnormally quick vocal fatigue. On examination, the characteristic cadaveric position of the cord was manifest despite the absence of any endolaryngeal lesion or pathologic condition of the neck or chest. But what was found in all these cases—and this sign has not been reported previously in the literature on the subject—was a slight pain which was produced on pressure between the trachea and the esophagus, that is, along the pathway of the inferior laryngeal nerve. This response to pressure by an inflamed nerve is, in my estimation, the pathognomonic sign of this disease.

Some interesting conclusions can be drawn from this clinical picture. The Semon-Rosenbach law states that slowly produced injury to the inferior laryngeal nerve will affect the abductor fibers primarily and the adductor fibers secondarily. Thus, for example, when a slowly developing aortic aneurysm starts to press on a recurrent laryngeal nerve, in the early stages the glottis closes normally but the left vocal cord abducts poorly. Later, when the adductor fibers are injured by the persistent and increasing pressure, adduction as well as abduction is impaired. Finally, with continued insult, the cord assumes the "cadaveric" fixed position midway between maximum abduction and adduction and is then considered totally paralyzed. Many explanations have been offered for the Semon phenomenon, the most probable being that the abductor fibers are weaker and respond more readily to abuse than the sturdier adductor fibers.

This clinical course has been observed regularly and accepted universally. The progressively developing paralysis has been noted innumerable times in cases of advancing pressure on the inferior laryngeal nerves. But in the present cases there was a characteristic difference in that the damage seemed to attack every fiber of the nerve simultaneously, abductors and adductors, producing a "cadaveric" cord from the very onset. Thus, Semon's law did not operate in the present cases of neuritis of the inferior laryngeal nerve, the condition resulting in "idiopathic" paralysis of the vocal cord. The prognosis was uniformly good. The patients recovered completely with no apparent deviation from normal of the movements of the cord.

A perusal of the literature discloses several reports of idiopathic paralysis of the vocal cord. Ellis and associates,[1] explained the lesion

1. Ellis, M.; Critchley, M., and Davis, E. D. D.: Discussion on Idiopathic Recurrent Laryngeal Nerve Palsies, Proc. Roy. Soc. Med. **39**:364 (May) 1946; J. Laryng. & Otol. **61**:286 (May) 1946.

on the basis of glandular dyscrasia and allergy; Goldsmith [2] reported a case of paralysis of the posterior cricoarytenoid muscle on a toxic basis following the administration of tetanus antitoxin; other writers [3] have noted paralysis following serum reactions, vitamin deficiencies (beriberi) or lead poisoning. In the discussion following the lecture of Ellis, Critchley and Davis on this subject at the Royal Society of Medicine, in London, Layton [4] reported a case of a paretic vocal cord in which the diagnosis had been "idiopathic paralysis," whereupon he had assumed that the vocal cord did not move because of ankylosis of the correlated cricoarytenoid articulation. Layton failed to describe the complete clinical picture of his case, but he erred if he intended to convey the impression that all idiopathic paralyses are the result of an ankylosed joint.

As mentioned previously, the immobilized cord in cricoarytenoid arthritis or perichondritis may readily be diagnosed by endolaryngeal inspection after one or several careful studies. This type of pathologic process differs completely from that of "idiopathic paralysis." If there is no difference between the right and the left cricoarytenoid area, the joint is not affected and the lesion lies definitely in the substance of the inferior laryngeal nerve. Finally, Layton offered a grave prognosis in these cases of paralysis on the mistaken premise that pathologic changes in the cricoarytenoid joint were responsible. It is known that the outlook is quite favorable in true cases of idiopathic paralysis.

I was frankly puzzled by the coincidence that in all 5 cases of idiopathic paralysis of the vocal cord the right cord was affected, and I can only offer some possibly explanatory observations (apart from that of the left laryngeal nerve's being better covered by the surrounding tissues). One patient presented lateral pharyngitis, decidedly more acute on the right side and accompanied with catarrh of the right eustachian tube, which may have indicated neuritis by propinquity of the right inferior laryngeal nerve. Another patient exhibited a pronounced difference in the degree of laryngotracheitis, the paresis of the right vocal cord coinciding with the far greater inflammation of the right side of the larynx and trachea. Incidentally, this patient had periostitis of the right side of the mandible shortly after the paretic right cord resumed normal function. A third patient, a dentist, who

2. Goldsmith, P. G.: A Consideration of the Medical and Surgical Problems Involved in Lesions Affecting the Recurrent Laryngeal Nerve, Tr. Am. Laryng., Rhin. & Otol. Soc. **50**:123, 1946.

3. Alcantara, V. C., and de Ocampo, G.: The Larynx in Infantile Beriberi, Arch. Otolaryng. **30**:389 (Sept.) 1939.

4. Layton, T. B., in discussion on Ellis, Critchley and Davis.[1]

was acutely allergic to procaine, had two attacks of idiopathic paralysis several years apart. In his case local care was supplemented by the use of antihistaminic drugs.

The history of the fourth patient is especially interesting. This man came under my observation after thorough examination at a laryngologic hospital, where he underwent many investigative procedures and was warned about the probability of an esophageal growth. My examination, including endoscopic and roentgenographic studies, indicated nothing pathologic. The patient responded rather promptly, within several weeks, to antineuritc medication combined with faradic stimulation, which was used in all the cases.

I have used the faradic method with much satisfaction. External electrodes are placed along the course of the recurrent laryngeal nerve, that is, in the zone between the pathways of the esophagus and trachea; internal electrodes are placed in the endolarynx. This is the method of choice, one which has afforded me palpable evidence of its efficacy as repeated laryngeal inspection indicated progressive improvement of the movements of the cords until complete cure was achieved. At the beginning improvement was very slow, but after a number of treatments a slight twitch of the cord indicated a definite turn for the better. The course of improvement was especially noticeable when I added the endolaryngeal electrodes to my equipment for faradic stimulation.

The fifth patient, the wife of a physician, came to me with total idiopathic paralysis of the right vocal cord. Her case was complicated by personal anxieties and shocks, which contributed to repeated relapses after satisfactory responses to the faradic treatment. At that, she responded remarkably to endolaryngeal treatment and obtained a strong voice after daily treatments. When she was discharged, the right cord moved normally in complete symmetry with the movements of the unaffected left cord.

In conclusion, the purpose of this paper is to focus the attention of the laryngologist on the occasional presence of "idiopathic paralyses" of the vocal cords. When both cricoarytenoid areas are normal, and when investigations of the neck and chest fail to explain a "cadaveric" cord, the laryngologist would do well to look for the pathognomonic tenderness on pressure deep in the neck in the area between the trachea and the esophagus. This sign alone will clarify the diagnosis and eliminate wasteful investigations. Fortunately, too, patients with this condition will respond to persistent treatment.

101 East Seventy-Fourth Street.

HYDROGEN ION CONCENTRATION OF NASAL SECRETION IN SITU IN NEWBORN INFANTS

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IN PREVIOUS communications [1] we reported that (1) the p_H of nasal secretion in situ in clinically normal nasal passages of infants and children was found to range within the levels of 5.0 to 6.7, indicating a slightly acid state of secretion, and that (2) during the act of crying and hyperventilating the establishment of an alkaline nasal p_H was a dominant finding in infants and very young children.

In the present investigation, an unusual opportunity to study the p_H of nasal secretion in situ in a group of 13 newborn infants, ranging from 2 to 8 days in age, was provided by the nursery of a metropolitan hospital. The subjects were 7 male and 6 female infants. Although all 13 newborn infants had normal intranasal structures, 1 of the group had cerebral edema and another an occipital encephalocele. All the children were born by the vaginal route of healthy mothers, except for 1, who was delivered by cesarean section of an eclamptic mother.

The technical aspects of the determination of the p_H of the nasal secretions in the newborn infants were carried out under aseptic conditions by the adult participants—physicians, nurses and a technician. A specially constructed silver–silver chloride glass electrode, designed for study of the nasal reaction in newborn infants and produced according to specifications furnished to the manufacturer, was employed in conjunction with the Coleman electrometer. Because of the inherent difficulty of undertaking p_H readings on the nasal secretions of newborn infants over prolonged periods of time, three consecutive readings were taken at one-half minute intervals.

1. Fabricant, N. D., and Perlstein, M. A.: Hydrogen Ion Concentration of Nasal Secretion in Situ in Infants and in Children, with a Comment on p_H Values in Pediatric Nasal Medication, Arch. Otolaryng. **47**:765 (June) 1948; p_H of Nasal Secretion in Situ in Infants and in Children: Effect of Hyperventilation and Crying, ibid. **48**:67 (July) 1948.

Of the 13 subjects under study, all but 1 newborn infant engaged in the act of crying and hyperventilating (table). The p_H of nasal

Data on p_H of Nasal Secretion in Situ for 13 Newborn Infants

Patients	Age, Days	Sex	Weight at Birth	p_H of Nasal Secretion	Comment
1	8	F	6 lb. 10 oz.	6.00 6.25 6.10	
2	4	M	9½ lb.	7.0 7.2 7.1	While crying, hyperventilating
3	6	M	6 lb. 14½ oz.	7.1 7.0 7.0	While crying, hyperventilating
4 (with cerebral edema)	2	M	7 lb. 13 oz.	7.1 7.0 7.1	While crying, hyperventilating
5	2	M	6 lb. 15½ oz.	6.8 7.1 7.2	While crying, hyperventilating
6	7	F	6 lb. 7 oz.	6.7 6.7 6.9	While crying, hyperventilating
7	6	F	7 lb. 13½ oz.	7.1 6.9 7.2	While crying, hyperventilating
8 (with occipital encephalocele)	6	F	8 lb. 3 oz.	7.2 7.2 7.2	While crying, hyperventilating
9	5	F	7 lb. 2 oz.	7.0 6.9 6.9	While crying, hyperventilating
10 (born by cesarean section; mother eclamptic)	5	M	7 lb.	6.9 6.7 6.8	While crying, hyperventilating
11	5	F	7 lb. ½ oz.	6.9 6.8 6.7	While crying, hyperventilating
12	7	M	10 lb.	7.0 6.8 7.0	While crying, hyperventilating
13	6	M	8 lb.	6.7 6.8 7.0	While crying, hyperventilating

secretion in situ for this single noncrying exception was found to range from 6.00 to 6.25. For the 12 newborn infants who cried the p_H of the nasal secretion was found to range from 6.7 to 7.2.

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OSTEOMA OF THE FRONTAL SINUS

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IT IS not the purpose of this paper to present a comprehensive report on osteoma of the frontal sinuses, as most of the facts now known about this condition can readily be found in such excellent recent articles as those of Teed [1] and Dowling.[2] We also direct attention to the article by Handousa [3] because of its many clearcut illustrations, roentgenograms and succinct case reports. The article by Cushing,[4] although dealing primarily with orbitoethmoidal osteomas and their complications, had considerable influence on the subsequent surgical approach to osteomas of the frontal sinuses with intracranial complications. Conley's [5] paper is of interest because it was the first to call attention to the repair of cranial defects by tantalum implants following surgical removal of osteomas of the frontal sinus.

An estimation of the relative frequency of osteoma of the frontal sinus might be deduced from the number of cases reported by different authors (table), or from the number of articles appearing in the medical literature on this subject. While osteoma of the frontal sinus appears to be more frequently encountered now than formerly, still, so far as the individual practitioner is concerned, it must be considered uncommon. For the surgeon who is to perform the operation in his own case the condition becomes rare, for a considerable number of the patients seen require no surgical intervention.

From the Eye, Ear, Nose and Throat Section, Fitzsimons General Hospital, Denver.

1. Teed, W. R.: Primary Osteoma of the Frontal Sinus, Arch. Otolaryng. **33**:255-292 (Feb.) 1941.

2. Dowling, J. R.: Osteoma of the Frontal Sinus, Arch. Otolaryng. **41**:99-108 (Feb.) 1945.

3. Handousa, A. S.: Nasal Osteomata, J. Laryng. & Otol. **55**:197-211 (April) 1940.

4. Cushing, H.: Experiences with Orbital Ethmoidal Osteomata Having Intracranial Complications, with Report of Four Cases, Surg., Gynec. & Obst. **44**:721-742 (June) 1927.

5. Conley, J. J.: Removal of a Frontal Osteoma and Correction of the Defect with a Tantalum Implant, Arch. Otolaryng. **40**:295-298 (Oct.) 1944.

In 1941 Teed [1] collected 321 cases from the literature. His bibliography covers the references and citations, and of these, approximately one third of the originals were recorded in English. Since 1941 we have found 20 articles in the literature dealing exclusively with this subject, of which 10 are in English. Teed [1] stated that it was nearly one hundred and fifty years from the time of the recording of the first case of osteoma in the medical literature, by Veiga in 1586, until the next case was recorded, by Vallisnieri in 1733. From 1926 through 1935 there was an average of more than one author per year who not only reported 1 or more cases but made an attempt to collect or estimate the total number of cases that had been reported in the

Frequency of Occurrence of Osteoma of Frontal Sinus

Author	Institution	No. of Cases in Series	Period of Investigation	No. of Cases of Osteoma
Andrews [25].....	New York Eye and Ear Infirmary, New York Ophthalmological and Aural Institute and Manhattan Eye, Ear and Throat Hospital	429,000	Up to 1887	3
Faulkner [25].....	Manhattan Eye, Ear and Throat Hospital	?	1904-1924	3
Teed [1].....	St. Josephs Mercy Hospital, Ann Arbor, Mich.	48,000	1918-1939	2
Handousa [3].....	Kasr-El-Ainy Hospital, Cairo, Egypt	840,000	1933-1940	13
Childrey [8].....	San Francisco Practice	Patients given roentgenograms of sinues 3,510	1939	10
Dowling [2].....	United States Army General Hospital	Ear, nose and throat patients 7,129	1942-1945	5

literature up to the time of his own article. In spite of the frequent reports, no author had reported as many as 4 of his own cases until Carmody [6] did so in 1935. It would seem that Carmody's [6] article may be taken as a point of departure in the literature, for since its appearance there have been reported 30 cases by Sättler [7] (1938), 9 cases by Childrey [8] (1939), 13 cases by Handousa [3] (1940) and 5 cases by Dowling [2] (1945). The cases reported by Sättler [7] and Childrey [8] were based on reviews of roentgenologic films, and those by Handousa [3] were collected from reviews of hospital records.

6. Carmody, T. E.: Osteoma of the Nasal Accessory Sinuses, Ann. Otol., Rhin. & Laryng. **44**:626-643 (Sept.) 1935.

7. Sättler, A.: Osteome der Stirnhöhlen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:464-479, 1938.

8. Childrey, J. H.: Osteoma of the Sinuses, the Frontal and the Sphenoid Bone: Report of Fifteen Cases, Arch. Otolaryng. **30**:63-72 (July) 1939.

We believe that many of the smaller osteomas are not reported. We have seen 6 cases of osteoma of the frontal sinus at this hospital in the past six months while on the eye, ear, nose and throat service. Of these 6 osteomas, 3 were small and asymptomatic. Two of the patients with the smaller osteomas were told to report back in eight months. The patient with the smallest osteoma, which was about 1 cm. in diameter, was under treatment for bilateral chronic suppurative maxillary sinusitis. We had roentgenograms of the sinuses of this patient over a period of sixteen years, and during that time the osteoma had shown no variation in size or shape. No further comment will be made on the cases of the 3 smaller osteomas, but we shall report the others later. We shall also report a case in which operation had been performed for osteoma of the left frontal sinus at another institution nine years previously and in which we operated for obstruction of the left nasofrontal duct. The last case is interesting from the standpoint of the late results which may follow removal of osteoma of the frontal sinus.

The cause of osteoma is unknown, but of the various theories advanced, that of a new growth from an embryonal periosteal rest would appear to us to be the most acceptable. The number of cases associated with trauma has been sufficiently large that many authors have concluded that trauma may at least be a factor in stimulating rapidity of growth. Whether suppuration is significant in initiating or stimulating the growth is an unanswered question, but a fair percentage of osteomas are associated with suppuration. It is not known whether the purulent discharge is a primary, secondary or incidental factor.

The relative frequency of occurrence of osteomas as compared with that of other benign tumors of the nose and paranasal sinuses was given by Eggston and Wolff [9] as 50 per cent of the total. This is a higher estimate than that given by most authors. It is not uncommon for the frontal and ethmoid sinuses to be involved with the same osteoma concurrently. Ersner and Saltzman [10] reported a case with simultaneous involvement of all the sinuses and cited Pfeiffer and Weingarten as each having reported a similar case. The patient of Ersner and Saltzman [10] had an osteoma involving the right frontal sinus, the ethmoid labyrinth, the sphenoid sinus and the right maxillary sinus; the tumor weighed 70 Gm. Hanley [11] reported three sep-

9. Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose and Throat, Baltimore, Williams & Wilkins Company, 1947, p. 750.

10. Ersner, M. S., and Saltzman, M.: Osteoma of the Sinuses, Laryngoscope **48**:29-37 (Jan.) 1938.

11. Hanley, J. S.: Unusual Case of Osteoma of the Nasal Accessory Sinuses, Laryngoscope **54**:235-237 (May) 1944.

arate osteomas occurring at the same time in their patient, one being in each frontal sinus and one in the ethmoid sinus.

The frequency of complications in the 321 cases collected by Teed [1] was roughly as follows: mucocele, 12 per cent; pyocele, 10 per cent; brain abscess, 3 per cent, and pneumatocele, 2.5 per cent. In 1 case meningitis was a complication, and in 1 cerebrospinal rhinorrhea. A few cases have been reported in which the osteoma grew back and deeply invaded the brain substance. Campbell and Gottschalk [12] reported such a case in which a mucocele had extended into the lateral ventricle.

The relative frequency of occurrence of osteoma of the frontal sinus in comparison with osteoma of the other paranasal sinuses in the 458 cases collected by Malan [13] is as follows: frontal sinus, 39 per cent; ethmoid sinus, 24 per cent; maxillary sinus, 9 per cent; sphenoid sinus, 2 per cent; nasal cavity, 5 per cent; origin doubtful, 7 per cent, and first cavity involved uncertain, 6 per cent.

The ages were recorded in 250 cases and the sex in 265 of the 321 cases collected by Teed.[1] The distribution according to age was as follows: first decade, 0; second decade, 25 per cent; third decade, 37 per cent; fourth decade, 17 per cent; fifth decade, 10 per cent; sixth decade, 7 per cent; seventh decade, 3 per cent, and eighth decade, 0.4 per cent. The distribution according to sex was 65 per cent for males and 35 per cent for females.

Perhaps the most frequent site of attachment of the tumor is in the region of the junction of the ethmoid bones and the frontal sinus, although almost every area in the frontal sinus has been reported as the site of attachment. A few of the reported sites are the posterior plate, reported by Coates and Kraus [14]; the roof of the sinus, by Lang and Armour [15]; the upper lateral wall, by Goodyear [16]; the interfrontal septum, by Teed [1]; the extreme upper part of the frontal plate,

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12. Campbell, E. H., and Gottschalk, R. B.: Osteoma of Frontal Sinus and Penetration of Lateral Ventricle, with Intermittent Pneumocephalus, J. A. M. A. **111**:239-241 (July 16) 1938.

13. Malan, E.: Chirurgia degli osteomi delle cavità pneumatiche perifacciali (contributo anatomo-clinico), Arch. ital. di chir. **48**:1-124 (Jan.) 1938.

14. Coates, G. M., and Kraus, F.: Osteoma of Frontal Sinus, Ann. Otol., Rhin. & Laryng. **50**:450-457 (June) 1941.

15. Lang, W., and Armour, D.: Ivory Exostosis, Growing from the Roof of the Frontal Sinus into the Orbital and Cranial Cavities, Removed Through an Osteoplastic Opening in the Cranium by Mr. Donald Armour, Proc. Roy. Soc. Med. (Sect. Ophth.) **12**:16-19, 1919.

16. Goodyear, H. M.: Osteoma of the Frontal Sinus Extending into the Orbit and Anterior Cerebral Fossa, Laryngoscope **35**:751-753 (Oct.) 1925.

by Johnson [17]; the upper mesial corner of the frontal plate, by Proetz [18]; the inferior mesial portion of the frontal plate, by Conley [5]; the floor external to the nasofrontal duct, by Hastings [19]; the floor involving both nasofrontal ducts, by Hanley [11]; the nasal spine, by Handousa [3] and the infundibulum, by Barnhill.[20] It is not uncommon for the surgeon to be unable to identify the site of attachment, owing to the extension and overgrowth of the tumor. A few cases have been reported in which the osteoma became detached and was extruded, usually through the orbit, remained in situ or became a loose body in the sinus. Jeschek [21] reported an osteoma that dropped out of the nose.

The size and rate of growth vary considerably, but in general it can be said that the tumor is slow growing and that the rate of growth is usually faster in persons of the young age group. Thomas' [22] patient had the tumor removed, and nine months later the growth was larger than it had been at the time of the operation. Osteomas at times tend to recur after removal, and a case was reported in which the tumor was removed ten times, each time with recurrence. Some osteomas have been followed for years with little or no appreciable change in size. Dowling [2] reported an osteoma the size of a pea; Conley,[5] one the size of a goose egg, and Hagaman,[23] one the size of a baseball; Hoover and Horrax [24] stated that osteomas of the frontal sinus weighing 1 pound (453 Gm.) have been described.

The symptoms, diagnosis and pathology of this condition will not be considered here, as discussions of these aspects are readily available in most articles on this tumor.

The physician, we believe, is more interested in the experience of others in the surgical treatment, since this is the only method of therapy,

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17. Johnson, W. H.: Osteoma of the Frontal Sinus, Arch. Otolaryng. **38**:318-323 (Oct.) 1943.

18. Proetz, A. W.: Evaluation of the Displacement Method, with a Review of the Literature, Ann. Otol., Rhin. & Laryng. **46**:699-734 (Sept.) 1937.

19. Hastings, H.: An Osteoma of the Frontal Sinus, Ann. Surg. **41**:624 (April) 1905.

20. Barnhill, J. F.: Report of a Case of Large Osteoma Involving the Right Frontal Sinus and Uncovering the Adjacent Brain, Ann. Otol., Rhin. & Laryng. **27**:1115-1117 (Dec.) 1918.

21. Jeschek, J.: Ueber das Verhalten der Stirnhohle beim Osteom, Monatsschr. f. Ohrenh. **68**:1107-1111 (Sept.) 1934.

22. Thomas, G. F.: Report of a Case of Osteoma of the Frontal Sinuses, Am. J. Roentgenol. **5**:341-343 (July) 1918.

23. Hagaman, V.: Osteoma of Frontal Sinus, Mississippi Doctor **20**:209-211 (Oct.) 1942.

24. Hoover, W. B., and Horrax, G.: Osteomas of the Nasal Accessory Sinuses with Report of a Case Illustrating the Trans-Cranial Approach to Orbital Structures, Surg., Gynec. & Obst. **61**:821-826 (Dec.) 1935.

than he is in other phases of osteoma. Unfortunately, surgical treatment is probably the topic least discussed in most papers. As osteomas vary greatly in their attachments, position, size, configuration, bony consistency and secondary complications, such a discussion would be most useful. Carmody [6] probably had this in mind when he wrote, "Since no one operator has had a sufficiently large number of cases to evaluate various methods of surgical procedure, the method must depend on the particular case."

Since Cushing's [4] article in 1927, there seems to have been general agreement that in the removal of those tumors in which a dural tear is likely to result the best approach is from the dural side. Cushing [4] was not the first to use this approach, for it had been used in 1918 by Lang and Armour.[15] Cushing,[4] however, was the first to use a strip of fascia lata to cover the tear in the dura, and his article greatly popularized the intracranial approach. Conley [5] repaired the torn dura by covering it with a segment of the frontalis muscle. Coates and Kraus [14] used bone chips over the dura to act as a nidus for bony repair of the posterior plate of the frontal sinus.

The incision used should be such as to give adequate exposure for removal of the tumor and to leave as little postoperative deformity as possible. Although many different approaches have been used, the most frequent has been a curvilinear incision above, below or through the eyebrow, with and without extensions along the corresponding side of the nose, as generally used in the external radical frontoethmoidal sinusotomy. In figure 1 we have sketched some of the other approaches used by different surgeons. Incisions *A* and *B* can be used for the intracranial approach. Incision *C*, with the long vertical extension, was used for insertion of a vitallium® plate to repair postoperative deformity. Incisions *C*, *D* and *E* should give adequate exposure, when indicated, for either an osteoperiosteal flap or insertion of a metal plate to repair postoperative defects. Incision *E*, when it could be used, would probably result in less visible scarring.

The tumor has usually been attacked by chiseling to free, or partially free, it from its attachment and then rocking it loose with large rongeur forceps. Hoover and Horrax [24] used a bone drill to free the osteoma partially. If the tumor is cancellous at its attachment, a curet is useful to clean up the residual bone after the main part has been removed.

There has been a difference of opinion as to the method of dealing with the nasofrontal duct and with the mucosal lining of the sinus. Johnson [17] stated that one should not remove the lining of the sinus if it is not infected. He left a fenestrated no. 12 catheter in the nasofrontal duct to promote drainage of the sinus and to prevent infection

of the sinus from the nose. Conley,[5] in order to prevent infection of the sinus from the nose, plugged a strip of frontalis muscle into the nasofrontal duct and by electrodesiccation fixed it to the surrounding tissue. Coates and Kraus [14] packed the nasofrontal duct. Dowling [2] stated that the duct should be left alone. Regardless of the internal management of the sinus, many surgeons have left a drain in the dependent part of the sinus to drain it externally.

Dowling [2] has given a good preoperative regimen. He used sulfonamide drugs before and after operation. Streptomycin and penicillin are now available when indicated.

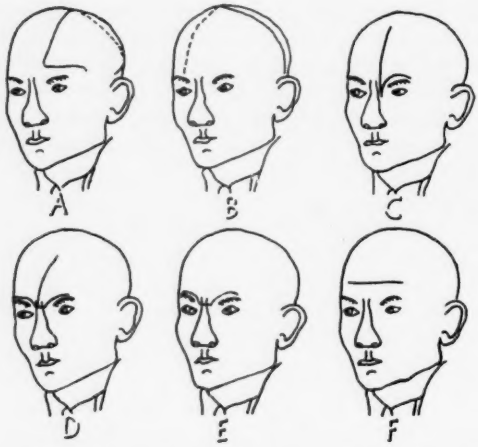

Fig. 1.—Incisions used by various authors in approach to osteoma of the frontal sinus: *A*, horseshoe temporal flap, Cushing [4]; *B*, transcranial post-hair-line flap, Hoover and Horrax [24]; *C*, curvilinear eyebrow-nasal incision with vertical extension, Conley [5]; *D*, eyebrow butterfly incision with vertical extension, Hagaman [23]; *E*, eyebrow butterfly incision, Hanley [11]; *F*, horizontal transfrontal incision, Dowling.[2] Note that the part of the incision shown above the eyebrow in *C*, *D* and *E* would ordinarily be made through the eyebrow to reduce visibility of the scar.

The operative mortality rate has been estimated by various authors as follows: Berlin,[25] in the preaseptic period, prior to 1880, as 25 per cent; Teed,[1] in the aseptic period, from 1875 to 1941, as 3.7 per cent, and Dowling,[2] in the period of biologicals, as less than 1 per cent. Teed [1] listed the causes of the postoperative deaths that occurred in 7 of 189 operative cases in the period from 1875 to 1941. The most frequent cause of death was meningitis.

25. Cited by Teed.[1]

REPORT OF CASES

CASE 1.—The patient, aged 22, a soldier of Latin-American descent, had lived all his life in Colorado. He had worked as a carpenter before joining the Army, four and one-half years prior to admission. He had never been in the tropics. He used no alcohol and smoked moderately. His previous personal history and family history were not significant. There was no history of trauma.

He was admitted to this hospital on May 11, 1948, with the complaint of having had a head cold for a week and a steady headache in the left frontal area for the previous forty-eight hours. He stated that he had not previously been treated for any trouble of the ears, nose or throat and had never experienced any pain

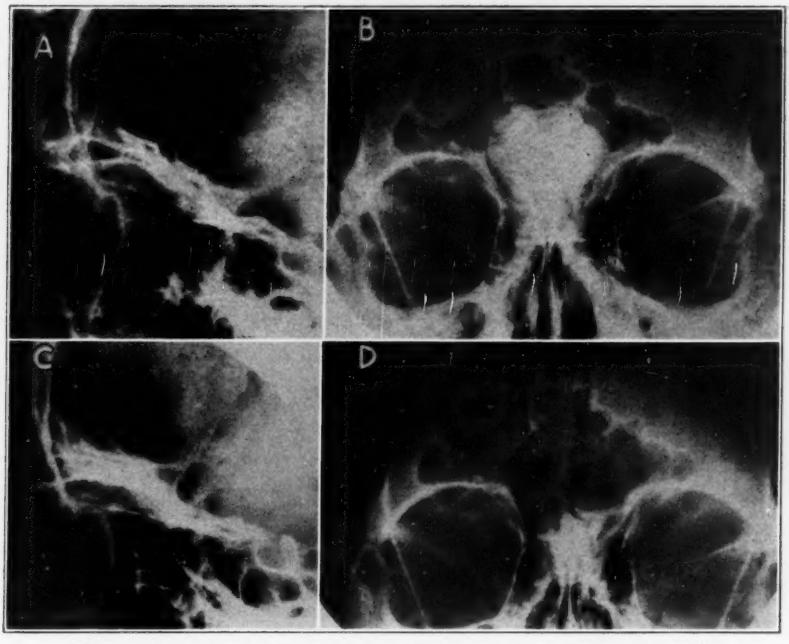

Fig. 2 (case 1).—*A* and *B*, lateral and anteroposterior roentgenograms taken prior to operation. A well demarcated area of increased density is present in the midline of the frontal sinuses. *C* and *D* lateral and anteroposterior roentgenograms taken after operation.

or tenderness in the sinal regions, or had prolonged headaches or any unusual amount of nasal or postnasal discharge. Examination of the sinuses in the eye, ear, nose and throat clinic showed moderate wet turgescence of the nasal turbinate bodies on both sides and pus under the left middle turbinate body. Transillumination showed darkening over the frontal area, so that an emergency roentgenogram of the sinuses was ordered.

The roentgenogram showed a well demarcated area of increased density in the midline of the frontal sinuses with a lobulated appearance typical of an osteoma

(fig. 2 *A* and *B*). The mass measured 2.3 by 3.2 cm. in the widest diameters. The lateral roentgenogram of the sinus showed pronounced thinning of the anterior plate of the sinuses, and the attachment of the osteoma appeared to be low on the posterior wall. The nasal accessory sinuses were otherwise normal in appearance.

Physical examination revealed an essentially normal condition except for the findings mentioned. The roentgenogram of the chest and the results of routine laboratory examinations, including serologic tests, were normal. Cultures of the pus under the left middle turbinate body were reported as showing Staphylococcus aureus, hemolytic streptococci and Aerobacter aerogenes.

The patient was placed on a regimen of therapy intended to clear up the suppuration prior to operation. The treatment consisted of vasoconstrictive nasal

Fig. 3 (case 1).—Osteoma after removal. (*1*) thinned-out anterior plate of the frontal sinus; (*2*) osteoma, from the frontal sinus; (*3*) portion of mucocele; (*4*) posterior plate of left frontal sinus.

tampons, nasal suction and injections of 50,000 units of penicillin intramuscularly every four hours for two weeks. Both maxillary sinuses were irrigated with an aqueous solution of 2,000 units of penicillin per cubic centimeter early in the period of hospitalization as a diagnostic measure to rule out any suppuration of the maxillary sinus. This was done in spite of the normal transillumination and the roentgenographic findings. The washings from both maxillary sinuses were clear.

The operation was performed on June 1, with the patient under anesthesia produced by intravenous injection of thiopental sodium. The butterfly incision through the eyebrows, as illustrated in *E* of figure 1, was used for the exposure

It was our intention to make an osteoperiosteal flap if this proved feasible, but the anterior plate of the sinuses over the tumor was of eggshell thinness, so that after the periosteum was elevated the thin anterior plate was chipped away. The osteoma was in the midline, having pushed the interfrontal septum into the lateral part of the right frontal sinus. The attachment, which was sessile and about 2 cm. in diameter, ran from the region of the left nasofrontal duct up the posterior wall of the sinus for about 2.5 cm. A mucocele was present in the lateral and superior portions of the left frontal sinus. The greater mass of the osteoma was removed by chiseling around its attachment until it could be rocked free (fig. 3). Piecemeal excision of the residual attachment was accomplished by chisel and curets. The interfrontal septum was removed with the osteoma (fig. 2*C* and *D*). The nasofrontal duct was left alone. The sinus was not obliterated, but the overhanging walls were smoothed. Closure was effected with black silk sutures, and a small rubber catheter was sutured into the lower part of the incision on the left side. The sinus was irrigated daily with an aqueous solution of penicillin, 2,000 units

Fig. 4 (case 1).—Postoperative appearance of the patient.

per cubic centimeter, for five days. Postoperative recovery was uneventful and the patient was asymptomatic. Two weeks after the operation he requested and was granted a furlough. He had only a moderate amount of noticeable deformity (fig. 4).

CASE 2.—The patient, an 18 year old white youth, reported to the eye, ear, nose and throat clinic on July 6, 1948, with the complaint of frontal headaches for the previous six months, which were present on waking in the morning and were usually gone by noon. He stated that the headaches appeared to make his vision "blurry." He also complained of stuffiness of the nose. Examination revealed nothing pathologic except for moderate deviation of the septum to the right. There was no nasal discharge. At this time a roentgenogram of the paranasal sinuses showed a smooth, rounded osteoma of the interfrontal septum, measuring about 2 cm. at its greatest diameter, with the main mass projecting into the right frontal sinus (fig. 5*A* and *B*). Because there was a possible rotary nystagmus on lateral gaze, neurologic consultation was requested, but no other significant neurologic

finding was noted. Results of the physical examination and of the laboratory work were not significant.

A submucous resection was done on July 21, and the postoperative course was uneventful. Because the persistent headaches were relieved only by constant use of tablets containing acetylsalicylic acid, acetophenetidin and codeine, it was thought advisable to remove the osteoma. This was done on August 12 (fig. 5 *C* and *D*).

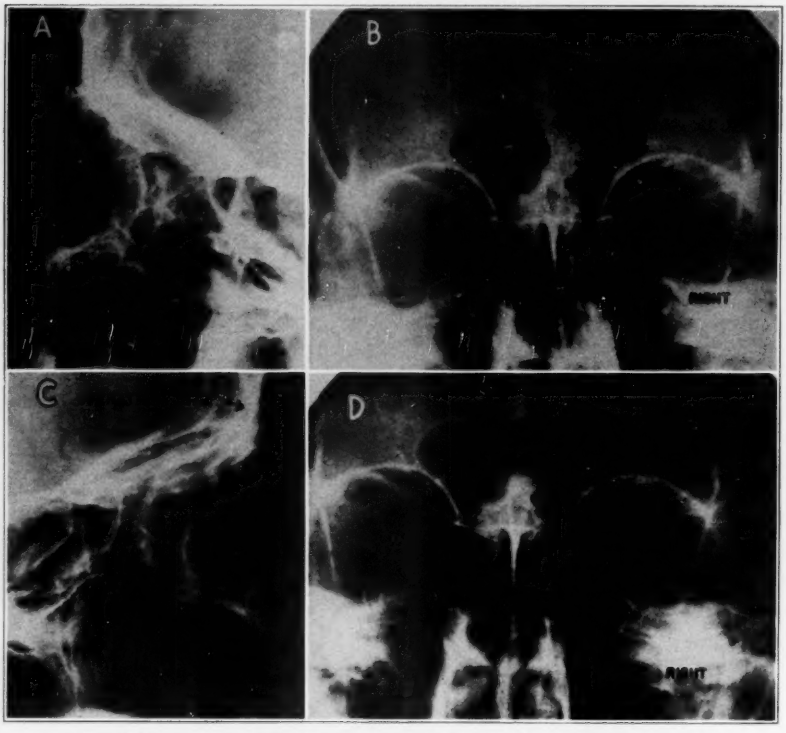

Fig. 5 (case 2).—*A* and *B*, lateral and anteroposterior roentgenograms taken prior to operation; *C* and *D*, lateral and anteroposterior roentgenograms taken after operation.

The operation was done with the patient under anesthesia produced by thiopental sodium, supplemented with nitrous oxide and oxygen. A horizontal frontal incision was made about 1 inch (2.5 cm.) above the brow line and extending about 1 inch to either side of the midline. The periosteum was elevated, and into each frontal sinus a small hole was chiseled sufficiently laterally to be outside the border of the growth, which saddled the interfrontal septum. These holes were enlarged superiorly and inferiorly in a semicircular manner so as to outline the extent of the tumor. The septum both above and below the osteoma was chiseled through

down to the posterior plate of the frontal bone. The interfrontal septum between the growth and the posterior plate was partially chiseled away. The osteoma was grasped with a large rongeur forceps and rocked loose from its base (fig. 6). The remains of the broad sessile attachment, which was about 1.5 cm. in width near the roof of the right frontal sinus was curetted away. The sinus was packed with petrolatum gauze, one end of which was left protruding from the left extremity of the incision wound. The mucosal lining of the sinus and the naso-frontal ducts were not disturbed. The periosteum was closed with 0 surgical gut U. S. P., and the skin, with black silk sutures.

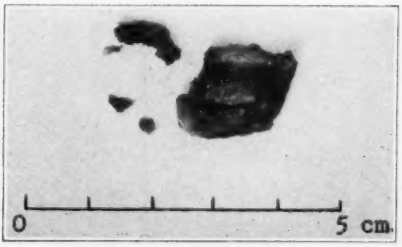

Fig. 6 (case 2).—Osteoma after removal.

Fig. 7 (case 2).—Postoperative appearance of the patient.

The postoperative recovery was uneventful. The patient was completely free from headaches and was discharged from the hospital approximately two weeks after the operation (fig. 7).

CASE 3.—The patient, a white soldier, aged 19, in 1946 had fallen on his face and fractured his nose. Since that time he had suffered from pain over the bridge of the nose and the left frontal sinus. A submucous resection was performed at a general hospital in the early part of 1947, but this did not relieve his symptoms. In June 1948 he was hospitalized one month because of pain over the left eye and a low grade fever. Treatment during this period consisted of nasal shrinkage and intramuscular administration of penicillin.

At the end of the last hospitalization, he was transferred to Fitzsimons General Hospital on July 26. Physical examination revealed an essentially normal condition except for impacted molar teeth. Roentgenograms showed a small osteoma, about 2 cm. in diameter, in the left frontal sinus and attached to the roof of the left orbit (fig. 8 *A* and *B*).

On August 27 the patient was given thiopental sodium (pentothal®) intravenously, and the osteoma was removed by external sinusotomy, the regular Killian incision being used. The tumor was attached to the roof of the left orbit by a broad base and to the posterior wall of the frontal sinus by a small pedicle. The patient was given penicillin intramuscularly for five days post-

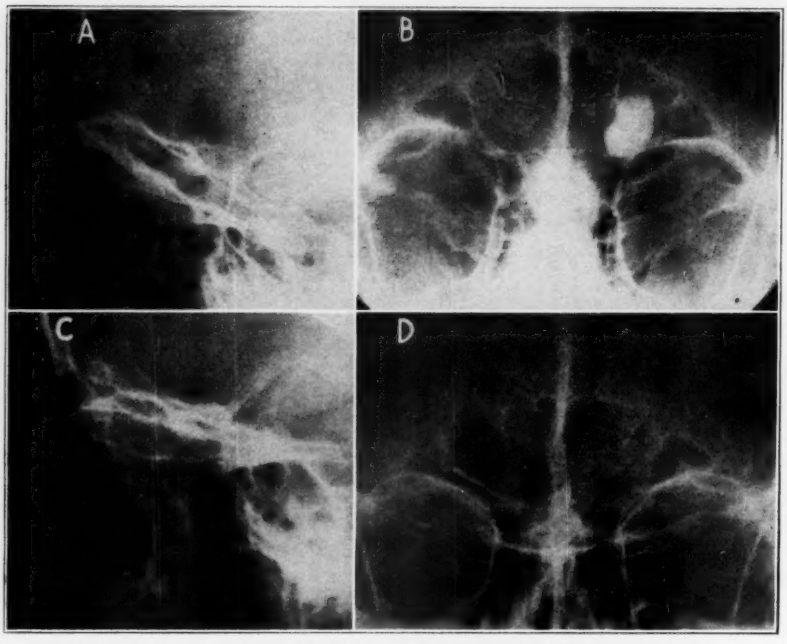

Fig. 8 (case 3).—*A* and *B*, lateral and anteroposterior roentgenograms taken prior to operation; *C* and *D*, postoperative roentgenograms.

operatively and made an uneventful recovery. One month after the operation he stated that he had had no recurrence of the symptoms. Figure 8 *C* and *D* show the postoperative roentgenograms. The tumor after removal and the patient two weeks after the operation are shown in figures 9 and 10.

Case 4.—We did not remove an osteoma in this case, but the patient had had such a growth removed from the left frontal sinus nine years previously, at a large, well known midwestern clinic. The history of the patient is interesting and instructive. He reported to the eye, ear, nose and throat clinic of Fitzsimons General Hospital on Aug. 14, 1948, with pain, tenderness and edema over the left frontal sinus. The left eye was bulging markedly; the lids of the same eye were swollen

to complete closure, except that the edematous palpebral conjunctiva of the lower lid was protruding about 1 cm. between them throughout the length of the lower lid. The periorbital tissues and the left side of the nose were inflamed, swollen and tender. No pus was seen in the nose. A scar from a previous Killian incision was visible in the left eyebrow. The eyegrounds were normal.

The history, obtained from both the patient and other clinics, was as follows: Sixteen years previously, in 1932, when the patient was 13 years old, he had fractured his nose in a boxing match. After this fracture he had an obstructed right nostril and pain over the left frontal sinus. In the following seven years he had had 7 intranasal operations, none of which had completely relieved the symptoms. The bridge of the nose had been depressed since the second operation.

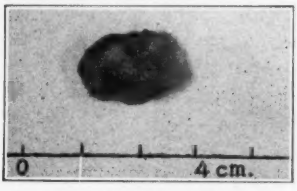

Fig. 9 (case 3).—Osteoma after removal.

Fig. 10 (case 3).—Appearance two weeks after operation.

He reported to the previously mentioned clinic in October 1939. Examination at that clinic revealed his nose to be filled with purulent secretion on the right side. A small window was present in the right antrum. The anterior tip of the right middle turbinate body had been removed. A partial submucous resection had been performed, leaving the entire palatine portion, which was deviated to the right. Roentgenograms showed a thickened membrane throughout the frontal sinus and a small osteoma of the left frontoethmoidal region (fig. 11 *A* and *B*). The submucous resection was completed on October 10. Two days later the left frontal sinus was entered through the external approach. The floor of the sinus was removed, and a spongy osteoma measuring 1.5 by 1 by 1 cm., was taken from the region of the nasofrontal duct, where it had blocked the duct. The frontal sinus above the osteoma was filled with a degenerated polypoid membrane, which was

curetted lightly but which was not completely removed. Anterior ethmoid cells were removed to fashion a large nasofrontal duct. A rubber tube drain was placed in the frontal duct and the wound drained.

The patient returned to the same clinic in May 1940 with the nasofrontal duct closed by scar tissue. The scar tissue was removed and the patient discharged. He returned again in January 1941 with the same complaint, and the left nasofrontal duct was again found to be closed. Detailed studies for extensive allergy gave negative results. Histamine desensitization was attempted, but the patient

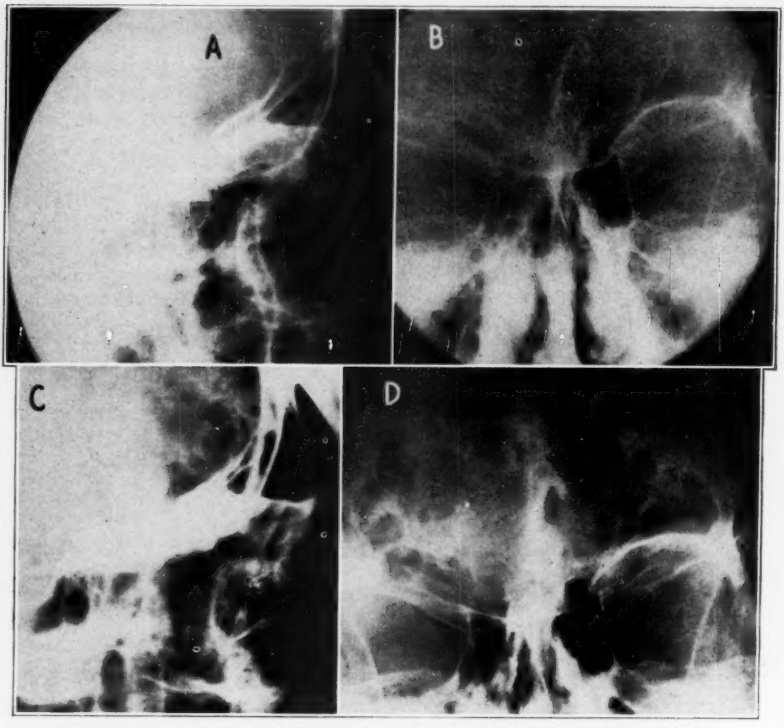

Fig. 11 (case 4).—*A* and *B*, lateral and anteroposterior roentgenograms taken prior to operation for osteoma; *C* and *D*, lateral and anteroposterior roentgenograms taken prior to operation for obstructed nasofrontal duct in the same patient.

did not remain long enough for treatment to be completed. In 1945 he returned to the clinic and had a rhinoplasty for the external nasal deformity.

In January 1948 he went to a well known, large eastern clinic because of a constant throbbing pain over the left eye and the left side of his head. The auriculotemporal nerve was divided at this clinic.

On May 5 he went to a hospital in the New England area with pain and swelling over the left eye. The upper lid was half closed and there were marked erythema and edema. Ocular muscle balance and visual fields were normal, and

there was no diplopia or conjunctivitis. The pupils reacted to light and in accommodation. The nasal mucosa of the left vestibule was greatly swollen, obstructing the airway in the region of the middle and inferior turbinate bodies. Physical examination showed an otherwise normal condition, and the laboratory studies were noncontributory. Roentgenograms showed pansinusitis, with absence of the roof of the left orbit as a result of previous operation. A radical operation was performed on the left frontal sinus, and a large mucocele was removed. A tantalum screen was inserted in the nasofrontal duct to provide constant drainage. The patient was discharged on June 5.

Approximately nine weeks after his discharge from the New England hospital, he was admitted to Fitzsimons General Hospital, with findings as previously described. Roentgenograms showed cloudiness of the left frontal sinus and the coiled tantalum wire mesh in the left nasofrontal duct (fig. 11 *C* and *D*). A diagnosis of suppurative inflammation of the left frontal sinus with secondary abscess in the left periorbital region was made. On August 18 a radical operation was performed on the left frontal sinus. There was pronounced hyperplasia of the sinus mucosa with granulation tissue. This was curetted out. The tantalum wire had acted as a scaffold across the nasofrontal duct, and its interstices were completely filled with granulation tissue. The wire and granulations were removed down into the left ethmoidal labyrinth. Pus was encountered in the periorbital tissue and in the posterolateral and anteromedial angles of the frontal sinus. A no. 16 French perforated catheter was left in the enlarged nasofrontal duct. The lids of the eye were sutured together to prevent erosion of the cornea and to afford protection to the protruding edematous conjunctiva. The patient was given intramuscular injections of penicillin and made an uneventful recovery. He was discharged from the hospital on September 18.

A NEW INTRALARYNGEAL APPROACH IN ARYTENOIDECTOMY IN BILATERAL ABDUCTOR PARALYSIS OF THE VOCAL CORDS

Report of Three Cases

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CINCINNATI

VARIOUS surgical procedures have been devised for the correction of bilateral abductor paralysis of the vocal cords. In almost all cases so reported the paralysis resulted from operations on the thyroid gland. No attempt will be made here to review all the procedures to be found in the literature, but those most commonly employed will be mentioned.

Surgical treatment of bilateral abductor paralysis of the vocal cords gained renewed interest after the report of King [1] in 1939. Originally he conceived the idea of transposing the anterior belly of the omohyoid muscle to the arytenoid cartilage, with the idea of restoring the function of the paralyzed posterior cricoarytenoid muscle. The operation, described as a means of opening the cord by use of the transposed muscle, actually was a new method of displacement of the cord.

Hoover [2] had previously reported success with submucous resection of the vocal cord. Kelly [3] described a procedure in which arytenoidectomy was carried out through a small window in the thyroid cartilage. Wright [4] presented a modification of Kelly's procedure in which he suggested that the extreme posterior end of the vocal cord be sutured to the external perichondrium at the lower margin of the window so as to

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From the Division of Otolaryngology, University of Cincinnati College of Medicine.

Presented with modifications before the Fifty-Third Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 13, 1948.

1. King, B. T.: A New and Function-Restoring Operation for Bilateral Cord Paralysis: Preliminary Report, J. A. M. A. **112**:814 (March 4) 1939.

2. Hoover, W. B.: Bilateral Abductor Paralysis: Operative Treatment by Submucous Resection of the Vocal Cords, Arch. Otolaryng. **15**:339 (March) 1932.

3. Kelly, J. D.: Surgical Treatment of Bilateral Paralysis of the Abductor Muscles of the Larynx, Tr. Am. Acad. Ophth. **45**:133, 1941.

4. Wright, E. S.: The Kelly Operation for Restoration of Laryngeal Function Following Bilateral Paralysis of the Vocal Cords: Report of Three Cases, Ann. Otol., Rhin. & Laryng. **52**:346, 1943.

obtain a larger glottic opening posteriorly. McCall and Gardiner [5] also presented a modification of the Kelly procedure for a more simplified operation in treating bilateral abductor paralysis of the vocal cords.

Orton [6] described a lateral transthyroid approach (Trotter's) in which an arytenoidectomy was performed after removal of the posterior third of the ala of the thyroid cartilage. The internal perichondrium and the thyroarytenoid muscle were then fixed to the external perichondrium and anterior portion of the split sternohyoid and thyrohyoid muscles.

INTRALARYNGEAL APPROACH THROUGH SUSPENSION LARYNGOSCOPE [7]

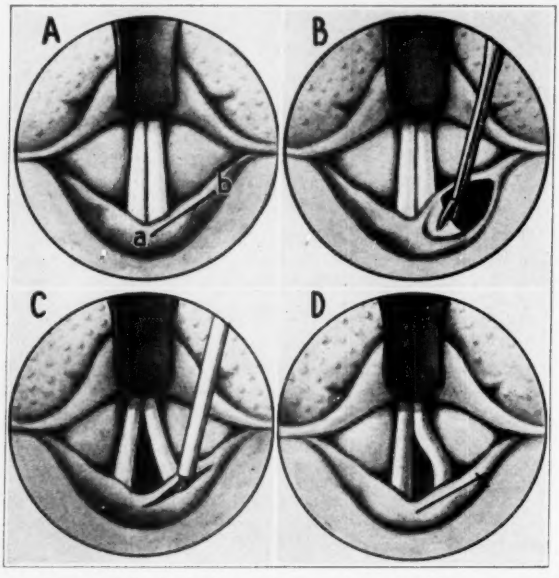
The patients selected in this instance for arytenoidectomy were those who had bilateral paralysis of the vocal cords as a result of operation on the thyroid. The removal of the arytenoid cartilage by the intralaryngeal approach is carried out through the Lynch laryngeal suspension apparatus, with combined local application of cocaine and intravenous injection of thiopental sodium U. S. P. (pentothal sodium®). The patient is hospitalized the day before operation. Preoperative medication is determined by the individual case. After the patient has been anesthetized, the suspension laryngoscope is placed in position, and further anesthesia is obtained with the local application of 5 per cent cocaine hydrochloride over the vocal cord and the aryepiglottic fold. A continuous supply of oxygen is administered through the tracheal cannula during the operation. An incision 1 cm. in length is made over the superior surface of the arytenoid cartilage and extended anterolaterally into the aryepiglottic fold (figure, *A*). The superior border of the cartilage is identified and tightly held by a grasping forceps (figure, *B*). This is more easily done if the forceps has a lock handle. Submucous dissection of the arytenoid cartilage is then carried out. The various muscular attachments are separated from the cartilage on its lateral aspect. The superior portion of the arytenoid cartilage is then rotated laterally so as to facilitate the separation of the muscular attachments on the medial aspect of the cartilage. A very sharp laryngeal knife is used to carry out the previously mentioned dissection. It is extremely important to avoid tearing or injuring the mucous membrane on the lateral wall of the larynx, since scarring in this area with a resulting cicatrix may nullify one's attempt to increase the glottic opening. After complete removal of the arytenoid cartilage, a guarded curved electrocautery point is deeply inserted through the incision along the

5. McCall, J. W., and Gardiner, F. S.: A Simplified Operation for Bilateral Abductor Paralysis, Laryngoscope **53**:307, 1943.

6. Orton, H. B.: Surgical Approach for Arytenoidectomy in Bilateral Abductor Paralysis of the Larynx, Laryngoscope **53**:709, 1943.

7. Thornell, W. C.: Intra-Laryngeal Approach for Arytenoidectomy in Bilateral Abductor Paralysis of the Vocal Cords: A Preliminary Report, Arch. Otolaryng. **47**:505 (April) 1948.

course of the thyroarytenoid muscle, beneath the vocal cord, and into the space previously occupied by the arytenoid cartilage (figure, *C*). Further lateral fixation of the posterior two thirds of the vocal cord is thus attempted by means of the contracture resulting from the electrocautery. Bleeding throughout the course of the operation is at a minimum and is readily controlled with the electrocautery unit. A chromic surgical gut suture was originally employed to close the anterior half of the incision, the posterior portion being allowed to remain open to afford drainage (figure, *D*). An acrylic obturator,



Schema showing (*A*) the incision *a-b* over the arytenoid area extending into the aryepiglottic fold; (*B*) position of the arytenoid cartilage, tightly held by a grasping forceps; (*C*) lateral displacement resulting from the removal of the arytenoid cartilage, with the guarded electrocautery point in position for cauterization; (*D*) lateral displacement of the posterior two thirds of the vocal cord, showing position of the susure at the anterolateral aspects of the incision.

fashioned to be inserted between the cord, is anchored inferiorly to the flange of the tracheal cannula and superiorly to the outer surface of the cheek with adhesive tape. This produces closer approximation of the edges of the wound and speeds primary healing. Laryngeal edema is pronounced for the first three to four days. The obturator is removed in three to seven days. The edema is noted to subside by the tenth to the fourteenth day. The cannula is removed twenty to thirty days after operation.

REPORT OF THREE CASES

CASE 1.[7]—A white woman aged 54 was seen in my office for the first time on Oct. 12, 1945. She presented a history of a thyroidectomy in September 1944. Immediately after the operation respiratory distress developed and required a tracheotomy. Her symptoms improved, and in four weeks the tracheotomy tube was removed. In March 1945 complete laryngeal obstruction developed, requiring replacement of the tracheotomy tube, which she retained up to the time I saw her.

On laryngeal examination, both vocal cords were found to be fixed in the midline position. A tracheotomy tube was in place. When the tracheotomy tube opening was covered, air could not pass through the glottic opening on inspiration, and both cords remained in their midline position. The remainder of the examination of the ears, nose and throat disclosed nothing of significance.

On May 4, 1947 the patient entered the hospital, and the following day arytenoidectomy was carried out on the right side through the Lynch suspension apparatus with the patient under anesthesia produced by intravenous injection of thiopental sodium, as I have outlined. The slight bleeding encountered was completely controlled by the electrocautery unit. The patient received injections of penicillin during her postoperative course. Thoracic pain delayed the patient's dismissal from the hospital. Physical examination and roentgenograms of the chest at this time revealed nothing abnormal. On May 14 she was dismissed from the hospital.

The patient was allowed to use her voice after the third postoperative day. Laryngeal edema subsided in ten days. The tracheotomy tube was completely sealed after three weeks and remained so until it was removed on June 23. The resulting glottic opening, when viewed indirectly, was 3 mm. posteriorly, the right vocal cord being retracted laterally.

On June 28 the patient was again hospitalized, and the tracheotomy opening was closed with use of local anesthesia. The patient was dismissed from the hospital on the following day. Her postoperative course was otherwise uneventful.

The patient has no difficulty with respiration at the time of this writing, and her voice is somewhat better than it was prior to operation. She has gained 25 pounds (11.3 Kg.) in weight since the operation.

CASE 2.—A white man aged 55 was first seen in my office on Dec. 8, 1947. His chief complaint was a history of shortness of breath on exertion and noisy respirations. The shortness of breath on exertion was so extreme as to prevent him from doing any strenuous work. The respiratory crow on inspiration became pronounced on exertion and was especially noticeable during sleep. The patient's neighbors complained of difficulty in sleeping in the summertime when the windows were opened because of this loud inspiratory crow.

He stated that in 1935 a thyroidectomy had been carried out elsewhere and was immediately followed by hoarseness. No respiratory distress was evident at that time. Two years later shortness of breath on exertion and an inspiratory crow, accentuated by exertion, were noted. The patient was forced to resign from his job and to limit himself to light work as a result of the shortness of breath. On approximately ten occasions laryngeal obstruction, accompanying an acute infection of the upper respiratory tract, became so evident that a tracheotomy was considered, but the patient was able to recover under conservative measures and close observation. At the time of my examination, both vocal cords were found to be fixed in the midline position, with only a glottic opening of 1 mm. A low transverse cervical thyroidectomy scar was noted. The remainder of the

examination revealed an essentially normal condition. The possibilities of further enlarging this glottic opening and establishing a better airway were thoroughly discussed with the patient, and he decided to proceed with the operation.

On Jan. 19, 1948 the patient entered the hospital, and the following day tracheotomy was carried out, with the use of local anesthesia. Immediately after this, arytenoidectomy was performed on the left side through the Lynch laryngeal suspension apparatus, as previously described. A modification of this procedure was carried out, in that placement of the suture at the anterior end of the incision was omitted and approximation of the edges of the mucous membrane was attempted by introducing a gold-plated O'Dwyer intubation tube into the glottis. The tube was not anchored inferiorly, and approximately six hours after operation the patient coughed it out. Penicillin was administered after operation. On the third postoperative day the patient had an acute infection of the upper respiratory tract, which prolonged his period of hospitalization to ten days. Laryngeal edema persisted ten days. The airway was found to be very adequate, and the tracheotomy tube was sealed over on the fourteenth day and removed on the eighteenth day. The patient had been instructed not to use his voice until the tracheotomy tube was sealed over. The resulting glottic opening, as viewed indirectly, was 4 mm. posteriorly. The left vocal cord was retracted laterally.

At the time of this writing, the patient has a very good airway, and his voice has shown slight improvement. Shortness of breath on exertion and the inspiratory crow have completely disappeared. The patient's activity has been considerably increased. He is now doing more strenuous work and has gained 15 pounds (6.8 Kg.) in weight.

Case 3.—A white woman aged 62 was first seen in my office on Feb. 14, 1948. She stated that in 1923 a thyroidectomy had been performed, with immediate resulting hoarseness. In October 1946 a second thyroidectomy was carried out. This was followed by complete aphonia, lasting three months, and hoarseness was noted after that. Respiratory distress on exertion was not noted until the following year. An inspiratory crow, especially noticeable at night, followed the second thyroidectomy. Since September 1947 any exertion had produced marked increase in respiratory effort and an increase in the inspiratory crow. In January 1948 a change in the voice was noted, the patient describing the voice as being more normal. Increased difficulty with respiration and increase in the inspiratory crow also occurred. On Jan. 22, 1948 a tracheotomy was performed because of almost complete laryngeal obstruction.

At the time of my examination the cords were found to be in the midline position with only a 1 mm. opening in the glottic space. When the tracheotomy tube was covered, the respiratory effort was greatly increased and an inspiratory crow was noted. The patient was hospitalized on March 1, 1948, and arytenoidectomy was carried out on the right side through the Lynch suspension apparatus, as previously described. The slight bleeding encountered was controlled with the electrocautery unit.

A modification of the procedure as mentioned in the report on case 2 was carried out: An O'Dwyer tube was placed between the vocal cords and anchored inferiorly to the tracheotomy tube and superiorly to the cheek. The tube was allowed to remain in position for four days. After this it was removed because of the marked edema noted around the tube. The patient was given injections of penicillin after operation. She was dismissed from the hospital on March 7. The tube was closed on the tenth postoperative day and was removed on the twenty-first postoperative day. The resulting glottic opening was 5 mm. The air-

way was quite adequate. The resulting voice was low in pitch and hoarse, but recently has shown some tendency toward improvement. She has had a 10 pound (4.5 Kg.) gain in weight since operation.

SUMMARY

A new intralaryngeal approach is presented for the correction of bilateral paralysis of the vocal cords which follows removal of the thyroid gland. The results have been excellent in establishing a normal airway in 3 patients so treated. In 2 patients the voice has shown a slight improvement. In 1 patient the resulting voice was poor but adequate and recently has shown some tendency toward improvement. Recordings of the voice were made before and after operation.

1002 Carew Tower.

THE ANTERIOR ETHMOIDAL NERVE SYNDROME

Referred Pain and Headache from the Lateral Nasal Wall

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THE ANTERIOR ethmoidal nerve syndrome is a name suggested for a series of symptoms resulting from irritation of the terminal branches of the anterior ethmoidal nerve. The referred pains arising from this nerve are chiefly of the sinus type but may also take the form of headache, sometimes of a migrainous character. Ephedrine applied to the anterior ethmoid fissure and/or the middle turbinate body has met with considerable success in the cure of the pain and headache of this origin. In April 1947, both anterior ethmoidal nerves were severed in a particularly stubborn case. The patient had not been able to carry on with her work, but since operation she has been rehabilitated. An effort will be made to apply recent physiologic discoveries in reporting the clinical course in this case.

To Littell [1] goes the credit for having cut the anterior ethmoidal nerve in 2 cases of this syndrome, with satisfactory results. His postulate that infection in the olfactory fissure (anterior ethmoidal foramen) is the fundamental cause of irritation of this nerve is only part of the explanation, in my opinion, as judged by considerable experience with this syndrome, extending over a period of fifteen years.[2]

Littell described and illustrated the gross anatomy of the anterior ethmoidal nerve from its exit in the orbit, its passage below the dura and its entrance into the anterior ethmoidal foramen and so into the nasal cavity, to become the nasal nerves. He has indicated how surface pressure on the mucous membrane covering the nerve can develop in this area of the olfactory fissure and that intranasal surgical treatment may suffice in many cases.

There is additional information which should be added to this discussion, and figure 1, taken from a previous paper,[3] illustrates bony

From the Department of Otolaryngology, Toronto General Hospital, University of Toronto.

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2. Burnham, H. H.: Headache from the Nasal Wall, Ann. Otol., Rhin. & Laryng. **46**:69, 1937.

3. Burnham, H. H.: An Anatomical Investigation of Blood Vessels of the Lateral Nasal Wall and Their Relation to Turbinates and Sinuses, J. Laryng. & Otol. **2**:569, 1935.

canals within the middle turbinate body into which the minute branches of blood vessels and nerves pass. It indicates where areas of potential pressure on nerves may take place from dilatation of surrounding veins, even though surface pressure might not be present.

REPORT OF A CASE

Miss C., aged 31, was first seen March 29, 1938. She complained of bilateral pain over the frontal, parietal, occipital and vertical areas. She experienced no pain behind the eyes, but had some aching and stiffness of the neck. She had

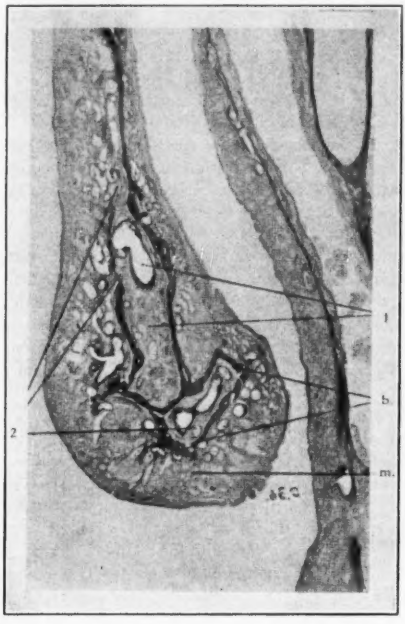

Fig. 1.—Cross section of middle turbinate body, showing the turbinate canal and vessels. *1*, is the middle turbinate artery and the plexus about it; *2*, various smaller canals which contain veins, these, in turn being continuous with vessels in the canal of the middle turbinate body; *b*, bone and *m*, mucoperiosteum; × 10. From Burnham.[3]

some pain every day, and frequently all day, for two weeks at a time, the pain even keeping her awake at night. There was stiffness of the muscles of the cheek and lower jaw when the pains were at all severe. The pain was at first a continuous ache, becoming throbbing as it grew severer and a sharp, stabbing pain occasionally when very severe. Her mother was subject to headaches.

Examination showed nothing of significance except for hypersensitive areas about the anterior ethmoidal foramen and over the course of the nasal nerves.

When this area was touched lightly with an applicator, she felt as though a red hot needle had been stuck into her nose at this point.

A 3 per cent solution of ephedrine sulfate was applied to this area, resulting in considerable amelioration of symptoms that day, but similar treatments twice a week for three months failed to stop the pain entirely.

At this time she was in a motor accident and had a fracture of the left malar bone, and I did not see her for several months. About ten months later I saw her again, as the old pain had once more been very severe for three weeks and she felt that the nasal treatment was the only thing that did any good. With the same form of treatment, there again was slow improvement. However, she stated that she had had a better summer than for some years.

A few weeks later war was declared, and I did not see her for seven years. In the interval she had joined the naval services as an officer, and one year before I saw her again she had had a return of the pain and headache and it had incapacitated her. No nasal cause had been found, and she had been turned over to the neurologic service, where her condition was diagnosed as psychosomatic; she was treated accordingly, but without improvement. When I saw her, she was delighted, as she felt that now her pain would be relieved. However, treatment once a week over many months gave no real improvement, and she was still incapacitated. I had felt for many years that an intractable case of this kind would justify the severing of the anterior ethmoidal nerve, but I had felt that the condition would have to be very bad indeed to justify such a procedure, as it was somewhat experimental. About this time I read Littell's article [1] and was greatly encouraged to try this operation. After the patient was discharged from the services, she came to my office and requested the operation.

On April 21, 1947 I severed both anterior ethmoidal nerves and vessels at their point of entrance into the anterior ethmoidal foramen from the orbital side. I varied Littell's technic by placing a small plug of periosteum over the foramen to prevent reunion of the nerve endings.

The immediate after-course was interesting. The patient was unable to take morphine, and so pantopon® (a mixture of the hydrochlorides of the opium alkaloids) was substituted. After operation there was considerable reaction in the soft orbital tissues, and she was unable to open her eyes for six days because of edema of the lids; she suffered very severe pain for three days. The pantopon® gave her so little relief that it was thought that it might as well be dispensed with. Codeine sulfate was used alone, with amobarbital sodium (amytal sodium®), ½ grain (31 mg.), given at night. At the end of two weeks she was feeling some relief. She was still getting ½ grain of codeine sulfate hypodermically four times daily and ½ grain amobarbital sodium at night. The pain was still worse than before operation, as the tissue reaction had not yet subsided.

Five weeks after operation she stated that she had begun to feel much better than before operation. Seven weeks after operation she said she had been free of pain over both malar areas for two weeks. The right frontal area was better than before operation, but the left, which had lagged about three weeks behind the right side, was about the same as before operation. She decided to drive her car north to the lakes the next day. She felt much encouraged, and her friends were pleased with her improvement. She could not read long at a time, as diplopia which followed operation was still present to a slight degree. The lagging of the left frontal area, as compared with the right, was probably due to a severer reaction of the soft tissues on this side and trauma of the supraorbital nerve from pressure of the retractor during the operation.

Postoperative Course.—September 30: The patient took a job in the summer and stated that her head was "an awful lot better." She still had pain in a narrow strip up the center of the forehead and about the scars. No pain was felt in the other areas except for two or three days at her menstrual periods. She stated that she had had less pain in the past one and a half months than since it began, twenty-one years before.

November 28: She had only a little soreness at the base of the nose; this was more or less constant, but was improving. After being out recently on a cold and windy day, she had had a severe pain up the center of her forehead to the hair line for two or three days. The drawn expression had gone from her face, although it had been noticeable to many of us who saw her before operation. The scars were barely visible.

Sensation. There had been astonishingly little loss of sensation as a result of the severing of the anterior ethmoidal nerves. The sensations of heat, cold, touch and tactile discrimination (sharp and dull) were now practically normal except in the area over the tip of the nose, in which sensibility was still dull, but was improving. The tip of the nose had felt cold at times, but this condition was improving.

Oct. 4, 1948 (one year five months after operation): She stated that the pain had been bad for two days in July. The weather had been particularly trying, and pain occurred during her menstrual period. However, the only pain she had felt was localized straight up the center of her forehead, with none at all in the face or anywhere else. Slight pain occurred at her menstrual period a month later, but, except for a slight occasional headache, "such as anyone might have," as she expressed it, she had been free from pain. Her family remarked on her great improvement and could not say enough about the benefit she had obtained.

Test of Sensation.—The tip of the nose still became cold, she stated. Occasionally a watery discharge occurred from the right nostril, but none from the left. The area about the scars was still tender, much as it was over a scar on her leg. An applicator applied to the nasal mucosa over the anterior half, i. e., the area normally supplied by the lateral nasal nerve, felt ticklish and caused her to sneeze, but, whereas this stimulus used greatly to aggravate the pain in her face and head, there was now no pain. The appearance of the mucosa of the turbinate body was slightly more intumescent than it had usually been before operation and gave the impression of the vessels being more dilated and relaxed. It shrank smartly after being touched with an applicator. Her nose felt normal and clear.

Comment.—The pain had practically stopped and was not stirred up by insertion of the applicator in her nose. She felt the stimulus as a dull sensation in her face, but not as pain.

The acuity of sensation of the skin of the bridge and the sides of her nose was still a little dull to sharp and dull stimuli, and that of the tip was slightly more so. The area in the posterior half of the nasal mucosa, supplied by the connection with the sphenopalatine ganglion, was hypersensitive and painful to the touch.

The sensation and reaction in the nasal mucosa usually supplied by the anterior ethmoidal nerve were normal; only the posterior area, supplied by the connections with the sphenopalatine ganglion, was hypersensitive.

GENERAL COMMENT

A discussion of the theoretic background of the anterior ethmoidal nerve syndrome is of interest. Three recent contributions to neurophysiology are applicable to this problem, and are mentioned in the order of their publication: (1) the work of Sir Thomas Lewis in demonstrating the role played by tissue metabolites in erythralgia, i. e., a burning and redness of the skin [4]; (2) the discoveries of Granit and his associates of the neurophysiologic department of the Karolinska Institute, Stockholm, Sweden, 1944,[5] and (3) Nathan's work on causalgia.[6]

1. In a paper published in 1937,[2] an effort was made to apply Lewis' theory of tissue metabolites to the problem of this particular pain and/or headache: (*a*) The irritation within the nasal mucosa from the products of bacterial invasion produces a hypersensitive or susceptible state. (*b*) As a result, substances (metabolites) normally produced in the tissues from external stimuli, such as changes of atmosphere and drafts, cause excessive stimulation of the neurovascular mechanism and sensory nerves. The headache or pain which results is in the nature of a referred pain and involves one or more branches of the trigeminal nerve. (*c*) The "tender spot" beneath the nasal bridge and in the area of the anterior ethmoidal foramen is believed to be due to pressure from congestion on the trunk of a hypersensitive nerve (nasal branches of the anterior ethmoidal nerve) in a bony foramen or canal, and this, in turn, may aggravate or produce the headache or pain.

2. In the work of Granit and his associates,[5] it is shown that severing of the sciatic nerve in an animal, or even pressure on it, will cause alteration of the action current in the nerve as far as 2 or 3 cm. proximal to the site of injury and that an odd reversal of currents takes place. Currents were found to pass from motor to sensory fibers of the nerve at this site and from sensory to motor fibers. A sensory to sensory fiber interaction was also demonstrated.

An application of this discovery to the present problem suggests that a traumatic condition of the anterior ethmoidal nerve may be set up by surface and/or intratissue pressure at the anterior ethmoidal foramen and the foramen of the middle turbinate body, causing interference with normal conduction of the action current, or, in other words, irritation of the nerve trunk for at least 2 or 3 cm. proximal to the site of trauma at the anterior ethmoidal foramen. If the anterior ethmoidal nerve be

4. Lewis, T., and Hess, W.: Pain Derived from the Skin and the Mechanics of Its Production, Clin. Sc. **4**:1, 1933.

5. Granit, R.; Leksell, L., and Skoglund, C. R.: Fibre Interaction in Injured or Compressed Region of Nerve, Brain **67**:125, 1944.

6. Nathan, P. W.: Pathogenesis of Causalgia in Peripheral Nerve Injuries, Brain, **70**:145, 1947.

traced back 2 or 3 cm. from the nasal cavity, the nasociliary nerve trunk is reached (fig. 2), and if the electric disturbance is continued farther back, many branches of this nerve are involved; still farther back the ophthalmic nerve trunk is involved.

Presumably, in Miss C.'s case most of the ophthalmic nerve was involved in this hypersensitive reaction, and when the pain was severe parts of the maxillary and mandibular divisions were affected, indicating involvement of the gasserian ganglion.

3. In Nathan's work [6] on causalgia, he advanced the hypothesis that pain is caused by efferent impulses in the sympathetic fibers of the nerve which stimulate the somatic afferent fibers at the point of damage, called the "artificial synapse." In other words, the outflow on the sympathetic fibers of the nerve may cause stimulation of the sensory nerve at the artificial synapse and cause pain referred to the area supplied by this nerve. The particular point of trauma from pressure (the artificial

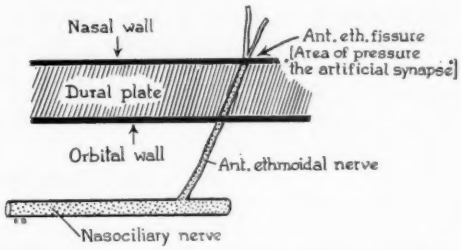

Fig. 2.—Diagram illustrating anterior ethmoidal nerve in relation to the surrounding area. The stippled area indicates the disturbed or hypersentitive area of the nerve as a result of pressure at the anterior ethmoid fissure and/or the areas of the middle turbinate body.

synapse) of the anterior ethmoidal nerve appears to be chiefly at the anterior ethmoidal foramen and, to some extent, over the bony canals within the anterior tip of the middle turbinate body. This nerve is presumably sensory, being a branch of the ophthalmic nerve, and contains autonomic fibers. The outflow from the autonomic nervous system would cause stimulation of the sensory nerve trunk at the artificial synapse. It may be postulated that the hypersensitive state of the nerve extends centrally and may involve branches of the nasociliary nerve and, still more centrally, the frontal and lacrimal branches of the ophthalmic nerve. This explains the pain in the malar area; occasional congestion of the conjunctiva; pain behind the eyeball, and pain in frontal and supraorbital areas, the root of the nose, the center of the forehead and the parietal area.

In the present case, the question arises as to why the patient occasionally had pain up the center of the forehead. This is the area supplied by the supratrochlear branch, which is therefore part of the ophthalmic nerve trunk from which the anterior ethmoidal nerve was given off. This nerve trunk was hypersensitive for many years, pain being touched off frequently by a hypersensitive anterior ethmoidal nerve. It is therefore to be expected that a terminal branch of this nerve may still have a low threshold for pain and that the stimulus producing pain in this nerve is due to a still hypersensitive nerve trunk rather than to a nerve center origin. The fact that it is giving less and less trouble would indicate that, with elimination of the trigger area, i. e., the anterior ethmoidal nerve, the nasociliary nerve connection is almost back to normal. The course of the illness in this case would indicate that the hypothesis of a hypersensitive nerve trunk, as indicated by physiologists, is a better explanation of the action of a trigger area than the irritation of nerve centers alone and that elimination of the trigger area will allow recovery of the whole nerve mechanism involved.

That a hypersensitive nerve was cut through explains why the pain in this case was worse immediately after sectioning and then gradually improved after the trigger area had been eliminated.

The history and course of the disturbance in this case emphasize the importance of locating a trigger area and eliminating it by treatment—medical if possible, surgical if necessary—rather than by treating the psychoneurosis alone. The latter aspect has been overemphasized in recent years. In a very high percentage of cases the anterior ethmoidal nerve syndrome will respond to conservative treatment, as outlined in "Headache from the Nasal Wall," and it is only rarely that drastic surgical intervention is necessary.

SUMMARY

The anterior ethmoidal nerve syndrome is a name suggested for a series of symptoms resulting from irritation of the terminal branches of the anterior ethmoidal nerve.

A case is described in which both anterior ethmoidal nerves were severed, with relief of the referred pain.

An explanation for this anterior ethmoidal nerve syndrome is suggested, based on neurophysiologic discoveries, the theory of tissue metabolites proposed by Lewis and the demonstration of nerve fiber interaction.

Medical Arts Building.

CHORDOMA OF THE MAXILLARY ANTRUM AND NARES

Report of a Case Clinically Resembling Hodgkin's Disease First Diagnosed by Biopsy of a Cervical Node

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CHORDOMAS in the maxillary antrums and nares are extremely rare. Rarer still are chordomatous metastases to cervical lymph nodes. Only 3 previous cases, those of Peters,[1] Potoschnig[2] and Lewis,[3] have been found with positive involvement of cervical nodes. Bailey and Bagdasar[4] thoroughly covered the history of chordoma from 1846 to 1929, and the origin and development of the notochord was aptly discussed anatomically by Huber.[5] The classification supplied by Coenen[6] is the most complete and satisfactory. This briefly recognizes three major groups: cranial, vertebral and sacrococcygeal. Under the first major cranial group, a fourth dental subgroup includes maxillary and mandibular chordomas.

Concerning biopsies, Adson, Kernohan and Woltman[7] stated:

> But biopsy, too, may present difficulties, as was experienced by Hirsch, from whose patient repeated specimens taken from a tumor in the tonsillar region resulted in such a wide variety of diagnoses and comments by eminent pathologists as almost to drive him to despair; a terse diagnosis of chordoma finally settled the

From the Departments of Otolaryngology, Surgery and Radiology of the Medical College of Virginia.

1. Peters, W.: Chordoma with Metastases, Deutsche Ztschr. f. Chir. **151**:191, 1919.

2. Potoschnig, G.: Ein Fall von malignem Chordom mit Metastasen, Beitr. z. path. Anat. u. z. allg. Path. **45**:356, 1919.

3. Lewis, N. D. C.: A Contribution to the Study of Tumors from the Primitive Notochord, Arch. Int. Med. **28**:434 (Oct.) 1921.

4. Bailey, P., and Bagdasar, D.: Intracranial Chordoblastoma, Am. J. Path. **5**:439, 1929.

5. Huber, G. C.: On the Anlage and Morphogenesis of the Chorda Dorsalis in Mammalia, in Particular the Guinea-Pig, Anat. Rec. **14**:217, 1918.

6. Coenen, H.: Das Chordom, Beitr. z. klin. Chir. **133**:1, 1925.

7. Adson, A. W.; Kernohan, J. W., and Woltman, H. W.: Cranial and Cervical Chordomas: Clinical and Histologic Study, Arch. Neurol. & Psychiat. **33**:247 (Feb.) 1935,

issue. Until the pathologist had been informed that the tumor extended into the retropharyngeal space, he had had, as may well be imagined, great mental hazards to surmount.

In another case they stated that ". . . in spite of the fact that the tumor had originated from the region of the spheno-occipital synchondrosis and had invaded the surrounding bone, little evidence of destruction of bone was seen in the postmortem roentgenogram."

Hass,[8] in 1934, collected 56 cases of chordoma of the cranial vault, the region of the cranium, the cervical portion of the spine and the nasopharynx and listed 77 pertinent references from the literature and added a very complete case report of his own. A history of trauma could be obtained in a high percentage of cases. The average age of the patients was 36 years. Pain in the neck or arms was almost invariably an early symptom. Bulbar symptoms, partial obstruction of the nasopharynx so that there was difficulty in breathing and various cranial nerve involvements followed from eight months to eighteen years after the onset of symptoms. In 2 instances, lateral extensions were palpable in the posterior cervical triangle. Two of his statements warrant quotation:

Furthermore, nasopharyngeal chordomas were described in patients who had no signs or symptoms referable to a tumor at the base of the brain.

Whenever anterior extension occurred it was customary to find ventral invasion of bone and soft tissues. The infiltration of bone which was usually symmetrical was not always restricted to the basisphenoid and the body and sinuses of the sphenoid bone. The regional petrous portions of the temporal bones, the ethmoid sinuses as far forward as the cristi galli and the osseous walls of the orbital cavities were involved . . . with ventral invasion into the walls of the nasal fossae and into the superior maxillary bones with the tumor encroaching on the nasal cavities, the maxillary antrums and the soft tissues of the face. . . . In one instance the temporal bone was penetrated by tumor which continued its invasion into the temporal muscle.

Owen, Hershey and Gurdjian[9] in 1932 reported the case of a 52 year old Italian cement worker with pain in his neck and both shoulders and arms, more pronounced on the left, involving the cervical portion of the spine. They detailed other case reports from the literature in 8 of which the tumor was believed to have originated in the cervical portion of the spine; in 2, from the odontoid process; in 1, from an intervertebral disk, and in 4, from vertebral bodies; in 2 the exact origin was unknown. They added:

In all cases that were examined by x-ray the diagnosis of tuberculosis was made although in our case secondary cancer was suspected. We believe from this

8. Hass, G. M.: Chordomas of the Cranium and Cervical Portion of the Spine: Review of the Literature with Report of a Case, Arch. Neurol. & Psychiat. **32**:300 (Aug.) 1934.

9. Owen, C. I.; Hershey, L. N., and Gurdjian, E. S.: Chordoma Dorsalis of Cervical Spine, Am. J. Cancer **16**:830, 1932.

standpoint alone, that in the consideration of tuberculosis of the cervical spine, chordoma must always be kept in mind as a possibility. There seems to be no typical x-ray picture of chordoma in the cervical region.

In 3 cases, the tumor grew anteriorly; in 1 it grew in the lateral aspect of the neck, and in 2 a mass projected into the pharynx.

Fig. 1.—Low power view showing the schneiderian membrane with the mucous gland and the tumor pushing from underneath the membrane.

In 1938, Ridpath [10] found 12 cervical, 52 spheno-occipital (10 naso-pharyngeal) and 91 sacrococcygeal tumors in patients with an average age of 36 years. He reported 2 cases from the otolaryngologic point of view, that of a woman of 43 years and that of a man aged 38 years.

10. Ridpath, R. F.: Chordoma, with Report of Two Cases, Ann. Otol., Rhin. & Laryng. **47**:649, 1938.

Stewart and Morin [11] reported the case of a man aged 59 years with increased swelling of the throat and left side of the neck. A firm swelling behind the postpharyngeal wall from the level of the upper part of the larynx led to the removal of the growth through an external lateral incision. There was destruction of the third cervical vertebra.

Fig. 2.—A low power view showing tumor cells with inflammatory cells very close to the schneiderian membrane.

Cappell [12] studied chordomas of the vertebral column, thoroughly reviewed the literature and found 77 cases reported up to 1928; he added 3 more cases, 2 in the cervical and 1 in the thoracic region.

11. Stewart, M. J., and Morin, J. E.: Chordoma: A Review with Report of a New Sacrococcygeal Case, J. Path. & Bact. **29**:41, 1926.

12. Cappell, D. F.: Chordoma of the Vertebral Column, with Three New Cases, J. Path. & Bact. **31**:797, 1928.

Radiation therapy has almost universally proved to be unsatisfactory in chordomas of all regions. Daland [13] in 1919 treated a mass below the right mastoid process in a woman aged 30 years, first with two massive doses with a total of 5 roentgen treatments, and a seven month follow-up showed recurrence of the mass following surgical intervention plus roentgen therapy. Stewart and Morin [11] said that, "probably

Fig. 3.—Nests of tumor cells and discrete tumor cells bathed in the inflammatory exudate.

on account of the indolent, slowly growing character of these tumors, treatment by radiation is almost without effect." Dickson and Lamb [14] reported a 2 cm. tumor area in the sacrum of a 41 year old man in which incomplete removal was followed by 900 r and then 160 r repeated

13. Daland, E. M.: Chordoma, Boston M. & S. J. **180**:571, 1919.

14. Dickson, J. A., and Lamb, C. A.: Sacral Chordoma, Ann. Surg. **93**:857, 1931.

three times. They felt that roentgen treatment apparently had been successful in checking development of the neoplasm. No metastases were found. Hass [8] quoted Goerke, who stated that "a combination of roentgen and thorium therapy was thought to be of value in one instance." Owen, Hershey and Gurdjian [9] tried high voltage roentgen therapy and thought it showed little effect in the way of result. However, this observation was based on observations on 1 case after three months'

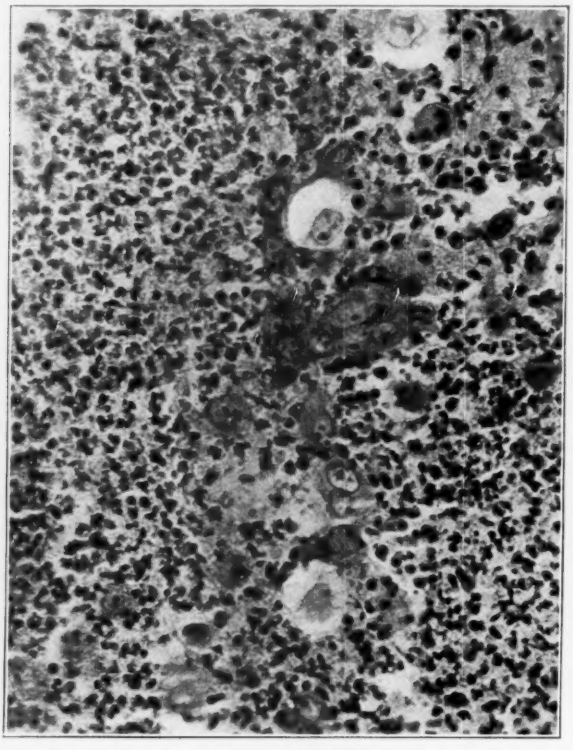

Fig. 4.—High power view showing nests of tumor cells, the most central cells of which are becoming vacuolated and ballooned (physaliferous).

follow-up. Adson, in discussing Van Wagenen's [15] paper, said, "It is my impression that all these tumors are malignant and some more malignant than others."

15. Adson, A. W., in discussion on Van Wagenen, W. P.: Chordoblastoma of the Basilar Plate of the Skull and Ecchordosis Physaliphora Spheno-Occipitalis: Suggestions for Diagnosis and Surgical Treatment, Arch. Neurol. & Psychiat. **34**:548 (Sept.) 1935.

Adams [16] recently reported a case of chordoma of the right frontal sinus in a 64 year old woman with dense sclerosis of the frontal bones around sinuses which were small but roentgenologically clear. Biopsy showed a cellular tumor with well defined cells with clear cytoplasm and small hyperchromatic nuclei. A 4,000 r surface dose to each of

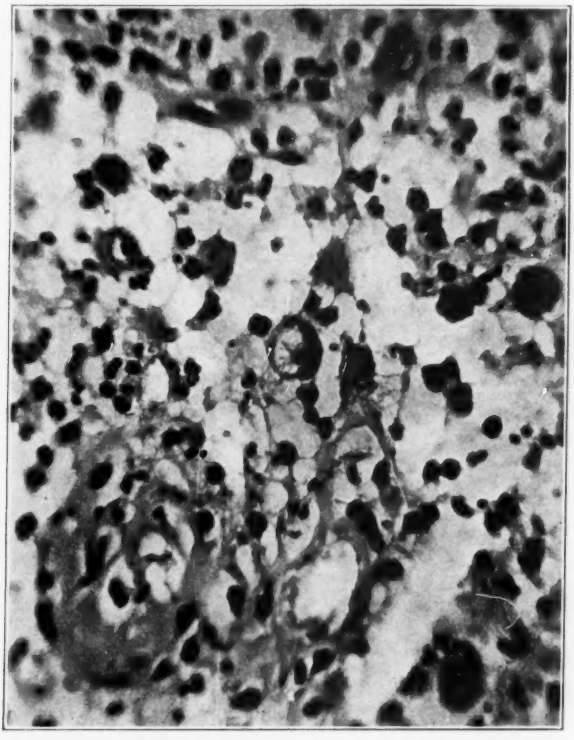

Fig. 5.—High power view showing the tumor cells undergoing mucoid degeneration. In the center of the field is one tumor cell with a large vacuole encroaching on the nucleus and producing a signet ring pattern.

two 5 cm. fields was followed by erosion of the floor of the right frontal sinus during the next six months. Biopsy of the frontal sinus membrane showed tumor still present. No metastases or extensions were present over one year later, and the operative site was healed.

16. Adams, W. S.: A Case of Chordoma of the Right Frontal Sinus, J. Laryng. & Otol. **62**:93, 1948.

Harvey and Dawson [17] noted that "the chordoma which had been described in the region of the maxillary antrum might possibly be confused with a parotid tumour but this is an unlikely site for the chordoma except as a late extension from the sella turcica."

REPORT OF CASE

A married white woman, aged 21 years, was first admitted to the Medical College of Virginia Hospital for a forceps delivery at term. The past history

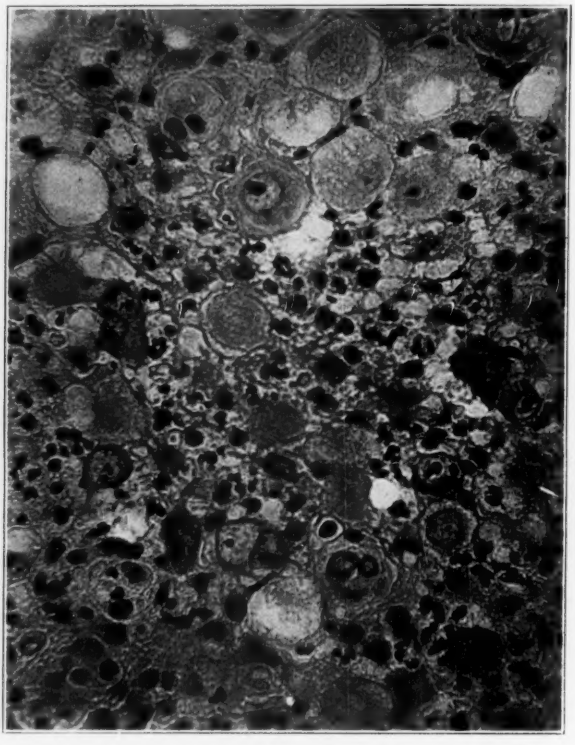

Fig. 6.—High power view showing multinucleated tumor cells and discrete tumor cells with a ring around them resembling plant cells.

showed measles, mumps, chickenpox, pneumonia and mastoiditis on the left, all early in childhood, before the age of 12 years. Tonsillectomy and adenoidectomy had been performed at the age of 12. The neck was free of palpable masses at that time. A routine flocculation test for syphilis elicited a negative reaction.

17. Harvey, W. F., and Dawson, E. K.: Chordoma, Edinburgh M. J. **48**:713, 1941.

The second admission was to the outpatient department ten years and three months later. The left ear had continued to discharge intermittently for twenty years. This discharge was usually more profuse during attacks of common colds. Two months before this admission she had noted pain and progressive swelling in the region of the left side of the neck, just under the mastoid process and behind the angle of the lower jaw. Her family physician had referred her to a surgeon, who had advised roentgen therapy, and four small doses of roentgen rays had been given at another hospital without obvious change in the size of the moderate-sized cervical mass.

Otolaryngologic examination showed the following positive findings at this time: There was an anterior superior perforation of the left tympanic membrane with slightly purulent drainage. The nasopharynx, nose and pharynx appeared perfectly clear. There was an irregular, nodular, firm, moderately fixed mass of nodes in the left cervical region just behind the angle of the jaw.

Roentgenograms of the mastoids in the Law and Towne positions showed chronic sclerotic changes in the left mastoid process. There were no erosions of the petrous ridges, sella turcica or foramen magnum or evidences of destructive or productive changes in the sphenoid and occipital bones.

After several days' observation, the 4 cm. mass in the left cervical region and a portion of the left sternocleidomastoid muscle and its sheath were removed by a general surgeon.

Adequate pathologic sections were examined by three pathologists, with one diagnosis "indeterminate," a second "metastatic carcinoma" and a third "Hodgkin's disease in which the granuloma has undergone a transition to sarcoma."

One month later a small, walnut-sized, mass appeared on the superior aspect of the left shoulder and three smaller nodules in the left preauricular, left posterior cervical and right midcervical regions, respectively.

Three months after the removal of the left cervical mass, for a period of fifteen months, with a clinical working diagnosis of Hodgkin's disease, based on the third microscopic diagnosis, and the lack of evidence of any site of primary carcinoma after numerous medical, neurosurgical and roentgen consultations, the patient received the following roentgen therapy:

Region	Date	Portal, Cm.	No. of Doses	Individual Dose, r (Air)	Total Dose, r
	Half-Value Layer, 0.9 Mm. Copper				
1. Left cervical	9/ 2/47- 9/22/47	10 × 15	8	150	1,200
2. Left outer supraclavicular	11/11/47-12/15/47	8 (circular)	10	200	1,950
			9	200	.....
3. Left inner supraclavicular	1/19/48- 2/23/48	8 (circular)	11	200	2,100
			10	200	.....
4. Left cervical	9/19/48- 5/ 3/48	10 × 15	8	200	1,600
	Half-Value Layer, 0.5 Mm. Copper				
5. Right cervical	7/22/48- 8/ 1/48	10 × 15	18	100-150	2,250
6. Left cervical	7/23/48- 8/ 1/48	10 × 15	18	100-150	2,250
7. Left axilla	10/15/48-10/18/48	10 × 15	3	250	750
	Half-Value Layer, 0.9 Mm. Copper				
8. Posterior thoracic spinal	10/16/48-10/18/48	20 × 8	2	200	400
9. Right lateral cervical	12/10/48-12/16/48	10 × 15	6	200	1,200

During this period the patient showed diminution in the size of the nodules treated, apparently an immediate response of the lymphoid tissue making up the bulk of the masses to the radiation. There was never complete disappearance of the nodelike areas. Pain and discomfort varied greatly in the left facial, cervical and shoulder regions. There was a loss of weight of 25 pounds (11.3 Kg.), and anorexia and nausea which occurred were attributed to the radiation therapy.

A third admission to the hospital one year after the removal of the cervical tumor mass was necessary for the performance of a therapeutic abortion in the sixth week of pregnancy.

A fourth admission was necessary three months later because of left nasal obstruction, severe pain, discomfort and swelling of the entire left side of the head and neck. Review of the sections from the cervical mass and lymph nodes by an experienced pathologist revealed "chordoma."

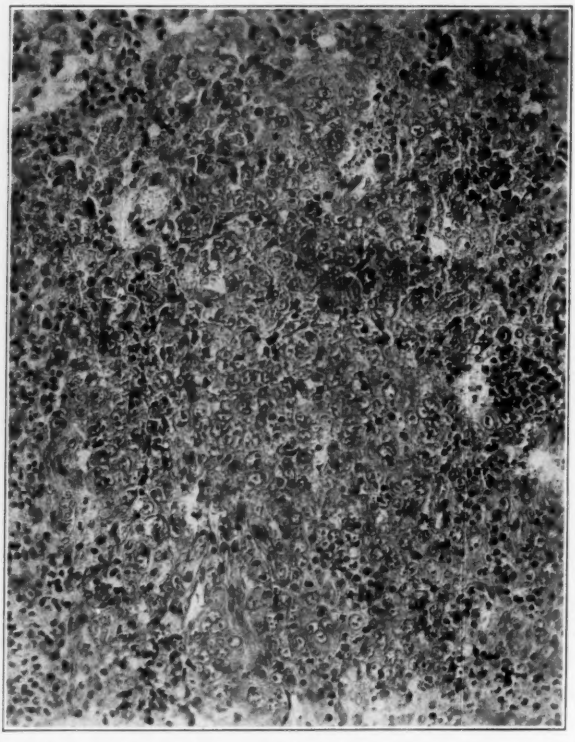

Fig. 7.—Medium power view showing a pattern similar to that of lymphoepithelioma.

Complete neurosurgical examination in the light of this diagnosis with roentgenograms of the skull, cervical thoracic and lumbosacral portions of the spine and chest showed no evidence of spheno-occipital, cervical or sacral chordoma. Roentgenograms of the nasal accessory sinuses showed complete clouding of the left maxillary antrum and densities in the left nares.

Otolaryngologic examination showed the same perforation of the left tympanic membrane with profuse foul drainage. Both nasal passages were obstructed, the turbinate tissue of the left side having a pale, blue, waterlogged appearance, with excessive tenacious polypoid plastic exudate completely filling the passage. The

nasopharynx was filled with this type of material, which seemed to originate in the left antrum.

On Oct. 16, 1948 the patient was given an intravenous dose of one of the nitrogen mustards and intramuscular injections of penicillin were started. Polypoid masses were removed from the left side of the nose and adjacent nasopharynx anteriorly. She was discharged from the hospital and followed in the outpatient clinic.

A fifth admission was necessary the following month because of severe pain in the left antral and cervical regions, and the neurosurgical department felt that the left gasserian ganglion was involved. On November 27 the otolaryngologic service performed a tympanomastoidectomy on the left (radical). Penicillin was given intramuscularly. The left nasopharynx and nasal passage was again obstructed, and polypoid masses appeared to have grown rapidly since the previous removal from the left antrum. The polypoid masses were made up of whitish gray material. The largest measured 1 cm. in its greatest diameter.

Microscopically the sections showed fragments of tissue of different types: 1. In some there was necrotic material with pus cells and hemorrhage. 2. In some there was vascular granulation tissue with leukocytes and tumor tissue. 3. In some there was normal schneiderian membrane (stratified columnar ciliated with stroma and glands) covering tumor tissue.

The structure of the tumor was very pleomorphic and to the casual observer confusing owing to the inflammatory process. The cell types presented (*a*) sheets of large cells with large vesicular nuclei, each containing two to three or more nucleoli; (*b*) syncytial masses with two to three or more nuclei; (*c*) cell nests in which the central cells were very large with foamy vacuolated cytoplasm (physaliferous); (*d*) cell nests in which the cells were very large with granular acidophilic cytoplasm. Some of these were beginning to become vacuolated, and some had become signet ring shaped. In these areas, there were large vacuolated masses, which had lost their nuclei. Here, coarse glycogen granules were demonstrable with Best's carmine stain. The granular nonvacuolated cells did not show glycogen granules. (*e*) Few mitotic figures were found, and these were in the granular cells. The pathologic diagnosis (P. F. S.) was chordoma.

Two months later the patient died, Jan. 14, 1949, in another local hospital, and necropsy was not obtained.

SUMMARY AND CONCLUSIONS

From the histologic picture presented in this case, in a 32 year old white woman, it becomes obvious that a review of the histogenesis of chordoma is indispensable for a differential diagnosis.

Chordomas originate from remnants of the primitive notochord. The primitive notochord is entodermal according to some and mesenchymal according to others. However, since there is no other tissue to behave like it and since it retains its individuality although it is closely enveloped and surrounded by mesenchymal structures, the concept of the French school as quoted by Harvey and Dawson,[17] that it originates *sui generis*, becomes very appropriate.

The tumor has many of the morphologic characteristics of cartilage, while it expands, infiltrates and even metastasizes like carcinoma. The tumor cells in certain instances may be large with clear cytoplasm

and distinct cell membranes and surrounded by hyaline and/or mucinous material and suggest the diagnosis of chondroma. However, other areas may show sheets of pavement cells, the most central of which are large and vacuolated (physaliferous) as seen in the illustrations. Some of the nests may be surrounded by hyaline bands. Mitotic figures are usually rare. This is the type which simulates carcinoma. When a chordoma of this type invades a lymphoid structure, as the nasopharynx or the cervical lymph nodes, the resulting picture may simulate a lympho-epithelioma or even Hodgkin's sarcoma. The differential diagnosis depends on the finding of the physaliferous cells and the vacuolated cells, as well as the paucity of mitotic figures, and clinically on its slow course and precarious response to irradiation therapy.

DENTAL CONSIDERATIONS IN FITTING HEARING AID EAR INSERTS

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ABOUT three quarters of a million hearing aids are sold annually in the United States, and the medical literature contains many articles concerned with the proper technic of fitting them. It is now generally agreed that if a hearing aid is to be used in aural rehabilitation it must be tailored to measure to give optimum results in each particular case.

An important part of this custom tailoring process is the fitting of the ear piece, or insert. In recent years it has been recognized that personal ear fittings must be used to get the best air conduction reception. Lederer and Hardy [1] found that a poorly fitting ear insert may lessen the efficiency of a vacuum tube hearing aid by as much as 20 decibels. They conclude: "Nor can too much emphasis be placed on the care with which the 'tailoring' of the ear mold is done." This is interesting, for it is commonly known that almost all the ear inserts sold are fitted by lay persons—hearing aid salesmen and saleswomen—who have no medical or dental background. It is our contention, based on facts to be presented, that even better results can be obtained if certain anatomic factors are taken into consideration before construction of the ear inserts.

REQUIREMENTS FOR A WELL FITTED EAR INSERT

The most important requirements that should be met by any well fitted ear insert are (1) that it should be comfortable and (2) that it should provide good acoustic seal; i. e., there should be no leakage or escape of sound around the insert.

The necessity for comfort is obvious. Wearing a hearing aid is in itself a bother to the average person, and an uncomfortable insert would become such an unbearable nuisance that the wearer would soon want

From the Hearing Clinic of the Eye, Ear, Nose and Throat Hospital.

1. Lederer, F. L., and Hardy, W. G.: Treatment and Training of the Hard of Hearing, Arch. Otolaryng. **43**:429-461 (May) 1946.

to discard the aid. We are of the opinion that buffing down rough edges and projections is not the complete answer to the problem, for often this is done with a sacrifice of snugness of fit and a loss of acoustic seal.

It is necessary to have the best acoustic seal possible, because leakage of sound decreases the efficiency of even the best aids. This decrease in efficiency is brought about because with leakage there is a weakening of most of the sounds reaching the middle ear. To compensate for this weakening a higher acoustic output must be used, and the higher the output the less the sensitivity. Another result of leakage is the squeal of acoustic feed-back.

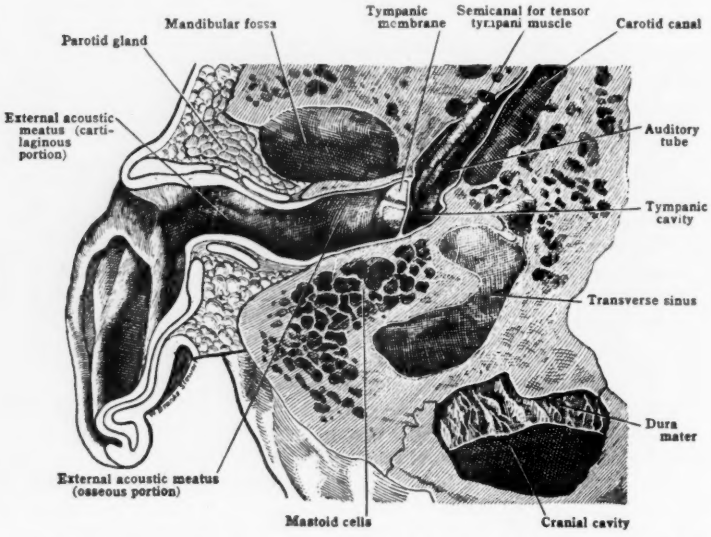

Fig. 1.—Horizontal section through the right ear showing close anatomic relation between the mandibular fossa and the mobile cartilaginomembranous portion of the external auditory canal. It is not difficult to see how abnormal condylar movements are transmitted to the mobile portion of the canal. (From Morris, H., and McMurrich, J. P.: Morris's Human Anatomy, ed. 10, Philadelphia, The Blakiston Company, 1942.

CONSIDERATIONS IN FITTING INSERTS

Anatomic.—A consideration of the anatomy of the external auditory meatus into which the ear mold is inserted will reveal some interesting facts (fig. 1).

We shall concern ourselves principally with the outer one third of the external meatus. This portion is cartilaginomembranous and mobile. One can demonstrate mobility by placing the small finger in the external

auditory canal while going through the acts of opening and closing the jaws. This mobility is due to the anatomic relation of the external canal: The anterior wall is in relation with the temporomandibular articulation and with the parotid gland. The gland is also in relation with the floor of the canal. Movement of the mandibular condyle is transmitted through the gland to the cartilaginous portion of the canal, which is drawn forward when the mouth is opened and compressed when it is closed.

When the joint functions correctly, movement of the canal wall is at a minimum for the individual person. On the other hand, when there is retraction of the condyle of the mandible, i. e., when the condyle is pushed posteriorly, the movement of the canal wall may be considerable. Between optimum functioning of the joint and pronounced dysfunction are many stages with varying degrees of movement of the external canal. Needless to say, the more movement there is, the more difficult it will be to obtain comfort and good acoustic seal when fitting an ear insert. Therefore, for the best possible fit of an ear insert, retraction of the mandibular condyle should be looked for, and, if present, the causes should be removed so that the condyles may be placed in their correct anatomic positions in the joint.

Dental.—Retraction of the mandibular condyle is due most frequently to malocclusion of the teeth, i. e., abnormality of the contact relation of their biting surfaces. Because, as Goodfriend [2] pointed out, the various movements of the mandible start from and return to centric occlusion of the teeth, an abnormal centric occlusion causes abnormal movements of the mandible and mandibular joints. As previously mentioned, this abnormality is transmitted to the external auditory canal.

Probably the commonest cause of abnormalities of centric occlusion is loss of permanent teeth in various numbers with inadequate replacement. Thus there results a disturbance in the normal arrangement of the occlusal relation of the upper and lower teeth.

A consideration of the frequency of loss of teeth at various ages should prove interesting. Table 1 is a compilation of the percentage loss of teeth during the ages of 10 to 75, based on 9,450 cases studied by Brekhus.[3] It shows that at the age of 20 up to 47 per cent of the persons studied had lost the lower first molar. After 20, the loss is progressively rapider. Members of the dental profession know this tooth as the keystone of the dental arch, for it is the first permanent tooth to erupt and around it all the rest of the teeth are arranged. Although the first molar is very important, it is well also to consider the loss of

2. Goodfriend, D. J.: Deafness, Tinnitus, Vertigo and Neuralgia, Arch. Otolaryng. **46**:1-47 (July) 1947.

3. Brekhus, P. J.: The Loss of Human Teeth, Minneapolis, University of Minnesota Press, 1928.

other teeth, particularly after 40, the age after which most hearing aids are worn. Brekhus [3] presented also the percentages of edentulous mouths in men and women between the ages of 20 and 70 (table 2).

TABLE 1.*—*Relative Loss of Teeth, Expressed in Percentages, During Ages From 10 to 75 in Male and Female Patients*

		Ages													
Teeth Lost	Sex	10	15	20	25	30	35	40	45	50	55	60	65	70	75
Lower six.....	M	10	30	40	45	50	55	63	70	72	77	80	85	88	90
	F	15	35	47	57	65	72	75	78	82	83	86	88	92	92
Upper six.....	M	5	15	23	35	40	50	55	62	69	75	81	86	89	93
	F	5	15	23	35	40	56	67	72	78	80	85	88	92	93
Upper five.....	M	0	3	11	18	29	40	48	57	62	68	75	80	86	90
	F	0	3	14	30	41	55	65	72	78	82	85	90	92	93
Upper four.....	M	0	4	11	18	29	37	46	55	62	67	75	80	86	90
	F	0	3	14	27	40	55	63	73	77	82	84	88	91	94
Lower seven...	M	0	2	8	16	25	35	45	55	63	67	76	82	87	90
	F	0	3	17	25	39	50	62	70	76	80	85	88	92	95
Upper seven....	M	0	0	3	12	21	33	44	60	64	73	79	83	90	93
	F	0	1	10	20	32	45	58	68	73	78	82	88	92	93
Lower five.....	M	0	3	9	13	19	26	33	41	49	56	64	70	75	79
	F	0	3	12	22	30	42	50	60	65	72	76	80	86	90
Upper two.....	M	0	2	6	10	20	30	39	48	55	62	69	74	81	85
	F	0	2	13	18	33	41	53	63	69	75	80	86	89	96
Upper one.....	M	0	0	2	10	18	25	35	44	51	60	68	75	82	89
	F	0	2	10	18	29	40	50	60	67	75	79	83	89	94
Upper three....	M	0	0	½	6	14	22	32	45	49	58	66	75	82	88
	F	0	0	6	15	26	40	50	60	67	72	78	81	87	95
Lower four....	M	0	0	1	6	10	15	23	30	40	47	58	64	70	73
	F	0	2	4	9	17	26	36	45	54	64	70	75	82	86
Lower one.....	M	0	0	1	2	6	12	20	28	39	48	58	65	72	76
	F	0	0	3	6	10	17	26	33	43	50	60	68	78	86
Lower two.....	M	0	0	¼	1	6	12	20	28	39	48	58	62	70	74
	F	0	0	2	4	10	16	26	36	43	54	60	66	78	86
Lower three....	M	0	0	¼	1	4	7	15	23	33	42	53	60	66	69
	F	0	0	1	4	9	16	25	36	41	53	60	65	78	85

* From Brekhus.[3] The percentages were based on a series of 9,450 patients.

TABLE 2.*—*Percentages of Edentulous Mouths in a Series of Men and Women Between the Ages of 20 and 70*

	Men			Women		
Age	Upper Jaw	Lower Jaw	Entire Mouth	Upper Jaw	Lower Jaw	Entire Mouth
20.....	1	0	0	0	0	0
30.....	8	3	3	20	8	8
40.....	26	13	13	43	21	20
50.....	44	29	28	60	40	39
60.....	62	47	46	73	57	55
70.....	76	58	56	86	78	76

* From Brekhus.[3]

In such cases there is no occlusion, and those patients who wear hearing aids should receive particular attention, for even though they wear complete upper and lower dentures there may be a loss of vertical height or intermaxillary distance, causing retraction of the mandibular

condyle. This loss of vertical height may be due either to improper construction of the dentures or to alveolar resorption following the wearing of dentures that had been properly constructed when first applied.

DIAGNOSIS OF CONDYLAR RETRACTION

The diagnosis of condylar retraction is not difficult and has been thoroughly discussed by Goodfriend.[2]

The first procedure is to feel the movements of the condyles during the opening and closing of the jaws by placing the fingers over the joints. Normally the movements are smooth; abnormally, there is lateral displacement or an uneven movement. In the second step, the tips of

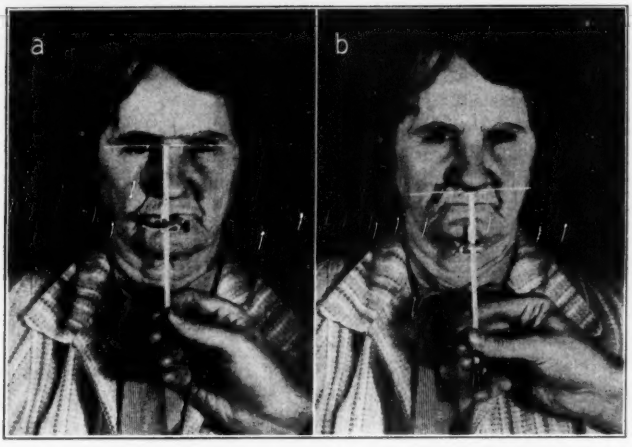

Fig. 2.—(*a*) Measurement A, the distance from the center of the pupil to the corner of the lip, normally should be the same as measurement B. In this patient, who has worn the same complete set of dentures for twenty years, A measured 71 mm. (*b*) Measurement B, distance from the base of the nose to the mental point of the chin, measured 58 mm.—a loss of 13 mm. in vertical height or intermaxillary distance. There was considerable movement of the outer third of the external auditory canals.

the little fingers are placed in the external auditory canals and the movements of the condyles are felt during speaking, swallowing, chewing, yawning, etc. When normally positioned, the condyles do not cause encroachment on the lumen and there is minimum movement of the external canal; abnormally, crepitus, jerky movements and encroachment on the canal are felt. This maneuver will also reveal inequality in movement of the two condyles.

The final procedure is to determine whether or not overclosure of the jaw with loss of vertical height or decrease in intermaxillary dis-

tance is present. This is done by comparing the measurement from the point of the pupil to the corner of the lip (measurement A) with that from the base of the nose to the mental point of the chin (measurement B). These measurements are illustrated in figure 2*a* and *b*. Normally, A equals B; with overclosure, A is greater than B. When the latter condition exists, there is said to be a loss of vertical height, a condition always accompanied with abnormal movements of the condyles and, hence, of the external auditory canal. The actual measurements may be made with a rule graduated in millimeters. We have found it convenient, however, to use the B-D facial surveyor[4] and feel that this is a more accurate method.

To determine the incidence of excessive movement in the external auditory meatus due to abnormal condylar motion, 100 hard of hearing patients were investigated. Forty-eight of these patients were wearing hearing aids, and 52 should have had them. Their ages ranged from 7 to 85 years, all but 12 being 35 years old or more. Using the diagnostic procedure previously outlined, we found that 80 per cent of the patients showed malocclusion with abnormal movement of the mandibular condyles. This condition was not taken into consideration, to the patient's knowledge, in a single case among the users of hearing aids when their ear inserts were being made. Sixty per cent of the hearing aid wearers were dissatisfied with the aids because of complaints that could possibly have been due to an ill fitting ear insert.

CONCLUSIONS

It is our contention, therefore, that if the best possible results are desired with a hearing aid an occlusal survey should be made. This means that there should be close cooperation between the otologist and the dentist in fitting ear inserts. When the difficulty is slight, there will be little need of corrective measures. On the other hand, such restorative measures should be seriously considered when there is marked encroachment on the canal; this is particularly important for those patients who are edentulous. If they were cared for, there would be fewer people who are forced to remove the ear insert of their hearing aid when eating or yawning because of the effects produced by improperly fitting dentures.

There is, we feel, still another benefit to be derived from restoring normal centric occlusion in many hard of hearing patients. This is in reference to the well established, but infrequently considered, part played by malocclusion in the production of conduction deafness. There is no agreement as to the exact pathogenesis of this condition, but there are many cases recorded attesting to the benefit derived from correction of

4. Manufactured by the Bee-Dee Distributors, New York.

the bite. In addition, we have in our files records of patients who have shown as much as a 25 decibel gain in audition from no treatment other than restoration of normal centric occlusion. Most of the patients requiring a hearing aid have either mixed or conduction deafness. If in a part of these persons deafness is based to some extent on abnormality of the temporomandibular joint, then it is logical to assume that once this difficulty is cared for there will be some lessening in the degree of their conduction deafness. If this is accomplished, the acoustic output of the hearing aid used could be lower. The tremendous benefit derived from lessening the acoustic output is obvious, for it is well known that the lower the acoustic output needed by the patient, the more satisfied he will be with the reception of the instrument.

SUMMARY

Although most hearing aid ear inserts are now individually fitted, insufficient consideration is given to the abnormal movement of the cartilaginous portion of the external auditory canal which may occur as a result of retraction of the mandibular condyles posteriorly.

The principal cause of abnormal condylar movement and position is malocclusion, the presence of which is not difficult to determine. That malocclusion is widespread in the population is evident from the results of a survey made by Brekhus on the loss of permanent teeth, the principal cause of malocclusion. This survey also revealed that in the age group in which most hearing aid users are found the incidence of malocclusion is greatest.

It is pointed out that if the hearing aid user is to get comfort and good acoustic seal with his ear insert, movements of the external auditory canal should be minimized. In 80 per cent of the cases studied this would have required a restoration of normal centric occlusion.

In addition to greater comfort and better acoustic seal, reestablishment of normal centric occlusion may cause some decrease in the conduction element of the deafness under consideration. With this decrease a lower acoustic output could be used and more satisfactory reception would result.

It is concluded that for the optimum fitting of hearing aid ear inserts cooperation of the otologist and the dentist is necessary in a large proportion of the cases.

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Case Reports

PLASMA CELL TUMOR SIMULATING BILATERAL MAXILLARY SINUSITIS

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Recognition of plasma cell tumors of the upper respiratory system and particularly of the accessory nasal sinuses has increased since attention has been called to their incidence by New,[1] Claiborn,[2] Jackson [3] and others. The otolaryngologist finds these tumors of particular interest because of their occurrence in the upper respiratory tract and, especially, in the maxillary sinuses. The plasma cell tumor is generally considered to arise from the bone marrow although many cases of extraosseous origin have been reported. The classic multiple myeloma is thought to be a disease of the bone marrow or of the blood-forming structures generally, and, while the plasma cell tumors seem to be a phase in the developmental history of multiple myeloma, the cause and nature of these tumors are not definitely known and their exact relationship to multiple myeloma [4] is not clear.

If the plasma cell tumor should be regarded as arising in the hemopoietic system, particularly the lymphocytes, such a premise might explain the occasional extraosseous occurrence of the tumor—in the soft tissues, such as the tongue, tonsils, pharynx and cheek.[5] To carry this premise further, later multiple involvement of marrow may represent a fundamental pathologic change in the hemopoietic system. Microscopically, the plasma cell tumor presents a predominance of plasma cells, with evidence of mitosis throughout the tissue.

In all instances of plasma cell tumors reported in literature, the symptoms associated with the tumors have varied with the location and activity. Thus, when the soft tissues of the face are involved, there is a gradually increasing swelling. In the oropharynx, the symptoms are referable to the act of swallowing; later in the progress of the disease, should there be invasion of the intracranial cavity diplopia and unilateral exophthalmos are evident. The subjective symptoms in the case herein presented were typical of chronic maxillary sinusitis. However, since the plasma cell tumor usually grows at a rapid pace, the progressive symptoms

From the Department of Otolaryngology, Gallinger Municipal Hospital.

1. New, G. B., and Harper, F. R.: Plasma Cell Myeloma of the Pharynx Without Sketetal Involvement, Arch. Otolaryng. **16**:50-54 (July) 1932.

2. Claiborn, L. N., and Ferris, H. W.: Plasma Cell Tumors of the Nasal and Naso-Pharyngeal Mucosa, Arch. Surg. **23**:477-499 (Sept.) 1931.

3. New, G. B., and Childrey, J. H.: Tumors of the Tonsil and Pharynx, Arch. Otolaryng. **14**:713-730 (Dec.) 1931.

4. Ewing, J.: Neoplastic Diseases: A Text-Book on Tumors, ed. 3, Philadelphia, W. B. Saunders Company, 1928, p. 420.

5. Figi, F. A.; Broders, A. C., and Havens, F. Z.: Plasma Cell Tumors of Upper Part of Respiratory Tract, Ann. Otol., Rhin. & Laryng. **54**:283-297 (June) 1945.

come on at an equally rapid rate, usually from six months to one year after the first early symptoms.[5]

It is further reported that in plasma cell tumors of the accessory sinuses roentgenographic studies reveal a more extensive involvement of bone than would be expected from the clinical examination; considerable erosion of the surrounding bony structures can be seen. This was so in the case here reported, both the lateral wall of the left orbit and the infraorbital plate being considerably involved.

In none of the reported cases have laboratory studies been of great value in early diagnosis of the plasma cell tumor. Presence of Bence-Jones protein in the urine is rarely reported in cases of plasmocytoma prior to development of the multiple osseous lesions, which is rather late in the course of the disease. Nor do the changes in the blood smear and alterations in the blood count serve as diagnostic aids from all reports, since both types of changes are slight.[5]

All authors are in accord that the plasma cell tumors are very sensitive to radiation and that once the diagnosis has been made irradiation is the treatment to be given. In fact, the disease has been reported held in check for many years with radiation therapy. However, it is warned that a guarded prognosis should be made, since there is always the possibility that the generalized multiple myeloma will arise at a later time; despite an apparent complete cure, the tumor must be regarded as highly malignant, and the patient should be kept under observation over a long period.[5]

The case to be reported illustrates many of the salient features of the plasma cell tumor previously discussed.

REPORT OF CASE

A Negro man, aged 60, was first seen in the outpatient clinic on Nov. 18, 1948, complaining of bilateral nasal obstruction, constant rhinorrhea and vasomotor symptoms. He stated that some two months earlier he had noticed nasal obstruction, first on one side and then on both sides; there was constant mucopurulent discharge, associated with a sensation of fulness in the antral region. There was no sinus pain. The left eye had been irritated for a period of three weeks. The patient did not complain of other physical ailments, and there had not been any loss of weight.

General physical examination revealed nothing significant except for pathologic conditions in the nose and throat. Examination of the nose revealed formation of polyps on both sides, so extensive that they protruded from the vestibule. The sinuses were not tender. A profuse mucopurulent discharge was evident in both nostrils. There was dacrocystitis on the left associated with the purulent conjunctivitis. The oropharynx and hypopharynx were normal. However, the nasopharynx when examined with a mirror showed what appeared to be polypoid tissue which filled the upper third of the space. The ears were normal except for moderate retraction and thickening of the tympanic membranes.

Roentgenologic study of the sinuses revealed definite and profuse haziness of both antrums. The remaining sinuses were essentially normal. On the basis of this diffuse haziness, irrigation was done; a greenish yellow purulent material was obtained. Since the patient did not show improvement, subjectively or objectively, after several irrigations, a study of the antrums was made after injection of iodized oil U. S. P.; each antrum accepted only 1 cc. of oil, and the subsequent roentgenogram revealed a marked filling defect suggestive of the presence of extreme polypoid degeneration or a tumor bilaterally.

Biopsy of the nasal polyps disclosed only the pathologic changes typical of a nasal polyp.

A bilateral Caldwell-Luc operation was performed. At the time of operation, the antrums were found completely filled with what appeared to be an organized necrotic inflammatory tissue which bled easily and which was firmly attached to the walls of the antrums but was not typical of antral polyps in appearance. In removing this tissue, a pathologic dehiscence was found in the nasoantral wall and in the floor of the orbit.

The results of microscopic study of this tissue were reported as follows: "There are groups of completely arranged cells which are irregularly trabeculated with narrow connective tissue bands and throughout which there are small fat cells. The medium-sized cells have a relatively large, usually round or ovoid nucleus, resembling plasma cells. Mitotic figures are seen throughout the slide."

The patient suffered no ill effects from the operation. He was discharged seven days later and was asked to return to the clinic within the second day; however, he did not return for three weeks. At that time examination disclosed a large mass on the right cheek. The left eye was proptosed and fixed. The patient complained of diplopia and blurring of vision. He had lost some weight.

Roentgen irradiation was begun; the total dose was 1,500 r to each antrum and 1,500 r each to the right and left zygomatic region. The patient made remarkable progress as a result of this treatment. The left eye returned to its normal position, and there was a return of function of the extraocular muscles. The plasma cell tumor tissue in the nasopharynx and in the nose itself seemed to have disappeared. Laboratory examinations revealed no abnormal conditions. Bence-Jones protein was not found. Roentgenograms of the long bones and scalp revealed no other lesions.

When the patient was seen June 1, 1949, there were no signs or symptoms of the disease.

COMMENT

The plasma cell tumor herein described is interesting in that it simulated a chronic maxillary sinusitis. The case shows the importance of careful microscopic study of all tissues removed at the time of operation, whether the tissue is thought to be a simple polyp or diseased tissue present in an antrum. The present case also serves to emphasize the occurrence of this neoplasm in the accessory sinuses, particularly in the antrums. Early diagnosis is important because of the efficacy of radiation in treatment of this type of tumor. However, from all reported instances, an over-all poor prognosis may be expected, since it has been found that even after an interval of seven to ten years the neoplasm may recur as multiple myeloma in other locations.

CONCLUSIONS

Plasma cell tumors of the maxillary sinuses are increasingly recognized, and an early diagnosis can be made; radiation therapy instituted in the early stages has brought remarkably quick improvement. But owing to the frequent development of multiple myelomas in later years, the prognosis can never be considered good. Pathologic studies of all tissues is of the utmost importance in diagnosing the plasma cell tumors.

RECOVERY IN OTITIC PNEUMOCOCCIC MENINGITIS

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A woman aged 55 retained good hearing practically unchanged after removal of the malleus, incus and drum membrane during the course of acute otitis media, mastoiditis and meningitis. She had been examined a month prior to her present illness, when her hearing for the whispered voice was within normal limits but audiometric examination showed moderate decibel loss bilaterally. Subsequent examinations were made with the same audiometer and under the same conditions.

The initial cell count of the spinal fluid was 9,000 per cubic millimeter. It is recognized that the higher the count and the older the patient, the less the chance for recovery.

The intracranial pathway of extension of the tympanomastoid infection was not demonstrable at the time of operation; it may have been through venous channels. Various routes of intracranial extension, such as via the subarcuate fossa from infected parantral cells, through the dura beneath the gasserian ganglion and from infected paratubal cells into the carotid canal, were shown by studies of serial horizontal sections of the middle ear and the mastoid in 26 cases of pneumococcic and in 13 cases of streptococcic meningitis occurring during the period from 1929 to 1932.[1] Although all deaths were proved to have resulted from meningitis secondary to the infection of the middle ear and the mastoid, the drum membranes had returned to an approximately normal macroscopic and microscopic picture, as shown in the photomicrographs and published cases.

Before the days of chemotherapy and use of the antibiotics, many patients with protective meningitis—that is, meningitis with no organisms demonstrable in the cerebrospinal fluid—were saved by drainage, removal of feeding foci and other measures. Rare was the patient with streptococcic infection—and rarer the patient with pneumococcic meningitis—who was saved.

The patient in the present report had had no previous aural infection to her knowledge. At the onset of her illness, she experienced a frontal headache for thirty-six hours and then became unconscious. On her entry to the hospital, she had been unconscious for seven hours. The right drum membrane was found red and bulging and was immediately incised, yielding serosanguineous exudate. The spinal fluid, with a count of 9,000 cells per cubic millimeter, yielded a pure culture of pneumococci. The first recorded temperature (rectal) was 105.5 F.; the white blood cell count was 12,900, with 43 per cent stab cells, 44 per cent segmented forms and 13 per cent lymphocytes. Daily laboratory examinations showed progressive improvement in the patient's condition. Two days after admission to the hospital she showed occasional rational response, and in four days she was fairly rational. Lumbar puncture, with intrathecal injection of 25,000 units of penicillin,

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From the Department of Otolaryngology, St. Louis University School of Medicine.

1. Boemer, L. C.: Some Observations on Temporal Bone Sections and Otitic Meningitis, Ann. Otol., Rhin. & Laryng. **42**:866, 1932.

was made twice daily for two days and then daily for ten subsequent days, with the intrathecal injection of penicillin omitted after the seventh day.

The temperature curve did not approximate normal until five days after tympanomastoid drainage (two weeks after the patient's admission to the hospital). Penicillin was given intramuscularly every two hours for four weeks, 500,000 units on admission, 100,000 units for two days and then 30,000 units until the day of the exploratory mastoidectomy, when the dose was increased to 100,000 units, with reduction to the preoperative dose in two days.

Sulfadiazine was given to maintain levels of from 8 to 13 mg. per hundred cubic centimeters. On the patient's fifteenth day in the hospital, leukopenia, with a count of 3,300 cells, developed. Sulfadiazine was discontinued, and in two days the white cell count had risen to 6,800. Lactated Ringer's solution U. S. P. with 5 per cent dextrose was given subcutaneously and intravenously in the amount of 5,400 cc. during the first eighteen hours and 3,000 cc. daily through the sixth day in the hospital, when the patient was able to take fluids by mouth. On her ninth day

Results of Audiometric Tests

	Right Ear			Left Ear	
Decibels	March 3, 1947	July 16, 1947	Nov. 15, 1948	March 5, 1947	Nov. 15, 1948
128	15	15	5	15	0
256	5	20	20	0	5
1024	20	20	15	5	5
2048	15	15	25	5	10
4096	25	25	55	5	10
8192	55	75	70	65	50
11584	Not heard at any examination date by either ear.				

in the hospital (two days after ventriculograms failed to show evidence of cerebral abscess), with slight tenderness and edema still remaining over the right mastoid bone, tympanomastoid operation was performed, with the patient under thiopental sodium (pentothal sodium®) anesthesia. The mastoid cells were soft in places and hemorrhagic throughout. Minimal granulations were present in the antrum and the aditus. The malleus and incus were removed and the drum membrane was excised subtotally, an anterior 2 mm. rim being permitted to remain.

The tympanomastoid cavity was completely epithelized and dry by the twentieth postoperative day. Hearing for the whispered voice approximated normal limits, between 20 and 40 feet (6 and 12 meters), and remains unchanged at the time of this report. Audiometric findings were not essentially changed by the operation or illness, considering the 10 decibel factor of error.

Comment.—It is considered that the rim of the drum membrane, permitted purposely to remain, grew together with connective tissue and squamous epithelium to cover the stapes in such a way as to permit fairly normal hearing.

Beaumont Medical Building.

Abstracts from Current Literature

Ear

USE OF NITROFURAN THERAPY IN EXTERNAL OTITIS. JACK R. ANDERSON and C. H. STEELE, Laryngoscope **58**:1279 (Dec.) 1948.

Most external otitis is of bacterial rather than of fungous origin. Nitrofurazone (furacin®) solution 1:500 is an effective germicide. After the ear has been thoroughly cleansed, the solution is placed on a cotton wick, which is inserted and left in place for twenty-four hours. The patient keeps the wick saturated with the solution by adding some periodically. If a fungous infection is suspected, a meta-cresylacetate-thymol solution is used also. Three treatments are all that are usually necessary, relief being obtained relatively quickly. HITSCHLER, Philadelphia.

PRACTICAL ASPECTS OF LABYRINTHINE TESTS. GORDON D. HOOPLE, Laryngoscope **59**:12 (Jan.) 1949.

Vertigo is becoming more common. To determine the cause is often difficult. A careful history and physical examination, including that of the hearing, are necessary, as well as labyrinthine tests. The combination of the three will give a reasonably good diagnosis.

The labyrinthine tests should be practical. Much detailed information is not to be anticipated from these, and such observations as can be made may be more confusing than helpful.

Various methods of performing these tests are discussed. The Hallpike-Cawthorne procedure is preferred. It is described in detail. It consists essentially in douching each ear with water slightly above and slightly below body temperature.

HITSCHLER, Philadelphia.

THE ROLE OF VIRUS INFECTIONS IN THE ETIOLOGY OF SEROUS OTITIS MEDIA. HAROLD G. TOBEY, Laryngoscope **59**:35 (Jan.) 1949.

The incidence of secretory otitis media has increased greatly in recent years. There must be other causes than that of mechanical obstruction of the eustachian tube; in fact, obstruction may have little to do with the condition. Politzer said that the discharging of fluid into the tympanic cavity was due mostly to inflammation of the mucous membrane of the middle ear. Some years ago such inflammation resulted in severe acute otitis media and often mastoiditis; now the insidious mild secretory otitis results. Chemotherapy cannot completely account for this change; rather, an avirulent bacterium or a virus is thought to be present.

Symptoms, signs and stages of the disease are described.

Politzerization may clear up the condition, or myringotomy, sometimes repeated, may be necessary. In some cases the cells of the mastoid process become involved, necessitating their removal.

The occurrence and importance of secretory otitis media should not be overlooked.

HITSCHLER, Philadelphia.

Pharynx

THE USE OF BISMUTH IN TONSILLITIS. W. STIRK ADAMS, Birmingham M. Rev. **15**:171 (Oct.) 1947.

Mr. Adams, Honorary Surgeon of the Ear, Nose and Throat Department, United Hospital and Children's Hospital, Birmingham, England, discusses the use of bismuth compounds in tonsillitis, reviewing the reports of some British and American authorities. He quotes J. F. Lewis, who stated that the organisms

etiologically responsible in acute follicular tonsillitis are fusiform bacilli and spirochetes, and not the streptococcal or hemolytic agents, as usually reported.

The author summarizes and concludes as follows:

"1.—A review of the original articles by Monteiro, and of published reports by all observers since, supports the conclusion that treatment by bismuth compounds controls the infection in acute non-specific tonsillitis and reduces the illness to a period of twenty-four hours in children and forty-eight hours in adults.

"2.—The disadvantages of intramuscular injections of bismuth salts are considered and rectal administration in suppository form is advocated.

"3.—The evidence for the etiological organism responsible for acute tonsillitis is considered, and on clinical grounds it is concluded that acute follicular tonsillitis is a spirochaetal disease.

"4.—The economic value of the treatment is evident and the discomfort to the patient of an injection is abolished."

The following doses are recommended for injection:

"Adult—Bis. sodii tartrate mgm. 25 (approx. 5/13 grain) . . . aq. sterile ad 1 cc.

"Infant—Bis. sodii tartrate mgm. 2.5 (approx. 5/130 grain) aq. ad 1 cc."

For rectal suppositories, this prescription is given:

"Adult—Bis. sodii tartrate mgm. 50 (approx. 5/6 grain) cocoa butter ad 1 gramme (15 grains)

or

bis. sodii tartrate mgm. 50, glyco-gelatin ad 1 gramme."

GORDON, Philadelphia.

Larynx

THE TREATMENT OF INTRINSIC CANCER OF THE LARYNX. JOHN D. KERNAN, New York State J. Med. **48**:178 (Jan. 15), 1948.

Dr. Kernan states that 95 per cent of intrinsic cancers of the larynx arise from the vocal cords. Satisfactory management calls for (1) complete removal of the primary growth and (2) removal or destruction of all lymphatic involvement. One has a choice of radiotherapy or surgery, or both. Radiotherapy destroys cells according to their sensitivity. Young cells and tissues in which the cells divide frequently are more sensitive to destruction than others. Too much radiation will, to be sure, destroy deep, resisting cells but will also cause the death of fairly normal cells. Dr. Kernan states: " . . . no radioresistant cancer, situated in a vital area, can be justifiably treated by radiotherapy."

Radiosensitivity and radiocurability are not always synonymous. Objections to radiotherapy are based on the fact that 96 per cent of these cancers are squamous celled and not commonly radiosensitive, and on the fact that if radiotherapy fails surgical treatment is difficult or impossible. These objections are no longer completely valid in these days of better radiotherapy, for if treatment is successful the patient has a healed larynx without deformity and a useful voice.

If surgical treatment is chosen, laryngofissure is the operation of choice for early intrinsic laryngeal cancer. The patient should be hospitalized a week or ten days preceding the operation, and preparation should be complete in every detail. To prevent postoperative infection, penicillin is given in doses of approximately 100,000 units daily during the preparation period. The choice of anesthesia should be left to an expert anesthetist. In Europe, local blocking of the cervical plexus is common; in America, most surgeons prefer general narcosis. In any case, the cough reflex must be kept intact, for hemorrhage and pneumonia are common complications. Kernan describes the methods of intralaryngeal removal originated by Lynch and continued by LeJeune.

Laryngofissure gives good results in early intrinsic cancer, that is, when there is mobility of the cords, no invasion of the arytenoid cartilages and no extension to the lymphatics. This operation is contraindicated when the ventricular band, the subglottic space or an arytenoid cartilage is involved. The operation is described in detail; it does not vary from the classic procedure, but various points are emphasized: (1) The interior of the trachea and larynx are anesthetized by the instillation of a few drops of 10 per cent solution of cocaine hydrochloride between the tracheal rings. [ABSTRACTER'S NOTE.—This is the so-called "tranquil tracheotomy" of Dr. St. Clair Thomson.] (2) Blood must be kept out of the lower airways. (3) "Excision of the growth is carried out so as to leave a surrounding free margin of tissue by making a straight vertical incision with scissors toward the arytenoid region above and below the growth, taking in, of course, the whole thickness of the tissue." (4) A bronchoscope is passed through the tracheotomy opening, and all fluid is removed. (5) The raw surfaces are powdered with sulfadiazine, and the soft parts are closed in layers.

Postoperative care calls for a semireclining or sitting position of the patient, retention of the tracheotomy tube for twelve to twenty-four hours, administration of sterile water by mouth for twenty-four hours, a fluid diet, gavage when necessary, continued administration of penicillin and removal of granulations as required. Healing takes place in two or three months. Total laryngectomy is reserved for invasion of the lymphatics or of parts external to the larynx.

Dr. Maurice Lenz speaks in the discussion of the joint observations which he and Dr. Kernan had carried on for seventeen years at the time of the report. Of 128 patients treated by roentgen therapy, 30 were free from clinical evidence of cancer for five to fourteen years afterward, but treatment failed in all 26 cases of cancer of the piriform sinus. Necrosis of cartilage was not unusual, but roentgen therapy "in early cancer of the cords gives as high a cure rate and a better voice than laryngofissure."

VOORHEES, New York.

NERVE BLOCK THERAPY FOR PAIN OF LARYNGEAL TUBERCULOSIS. E. M. PAPPER and E. A. ROVENSTINE, New York State J. Med. **48**:622 (March 15) 1948.

Working in the Department of Anesthesia, Bellevue Hospital, the authors treated 15 patients who had the distressing symptoms of dysphagia and exquisite pain.

Entrance is made in the midline just above the thyroid notch with the patient in a supine position, and the needle is advanced toward the hyoid cornu and stopped just below it, where infiltration is desired. Into the superior laryngeal nerve, which also contains some motor elements, 2 cc. of 2 per cent solution of procaine hydrochloride is injected. Ten minutes later, 2 cc. of absolute alcohol is instilled at this site. Immediate relief is obtained for weeks or months. Some 15 patients were thus treated with bilateral injections when pain was intense during deglutition and phonation. All these patients had advanced pulmonary tuberculosis, and all died later. The course of the disease was not altered by the procedure. Three patients had only partial relief; failures occurred in the treatment of 2 patients. Two required a second nerve block, but relief was gained at both sittings. Diagrammatic drawings illustrate the surgical anatomy and the method of injection.

(ABSTRACTER'S NOTE.—While such injections are not new in a broad sense the approach is different from that in ordinary use, in which the injection is made into the superior laryngeal nerve at the point where it passes over the thyroid cartilage into the larynx.)

VOORHEES, New York.

CARCINOMA OF THE ESOPHAGUS WITHOUT DYSPHAGIA. ABRAHAM W. FREIREICH, New York State J. Med. **48**:911 (April 15) 1948.

This report was made before the clinical conference of the Fourth Medical Division, Bellevue Hospital, Nov. 24, 1947. It concerns the case of a man of 79 with weakness, anorexia, an episode of alternating diarrhea and constipation and a weight loss of 25 pounds (11.3 Kg.) of two months' duration. An unusually complete physical examination was made; however, the patient was too severely ill for esophagoscopy. The gastrointestinal tract was studied only at each end. A roentgenogram of the chest showed widening of the supracardiac aorta. In the discussion, Dr. Emanuel Appelbaum states that dysphagia is early and persistent in cancer of the esophagus. Metastasis is frequent and is also early, but symptoms may be indefinite when no compression of the lumen is present. In fact, cases have been described in which cancer of the esophagus was determined only after death. The autopsy report, by Dr. Henry Spitz, describes the main lesion, at the level of the bifurcation of the trachea, as a fungating, ulcerated, circular mass; the lumen of the esophagus was about normal. Microscopic examination revealed a squamous cell carcinoma which had widely and diffusely invaded the external esophageal wall. This was an example of an occult condition with nothing specific in the history, physical findings or laboratory reports. "Even the terminal events are nonrevealing."

VOORHEES, New York.

Nose

REPAIR OF THE SEPTAL PERFORATION A RHINOLOGIC PROBLEM: A RHINOPLASTIC APPROACH (AUTHOR'S TECHNIQUE). ROBERT C. SEELEY, Laryngoscope **59**: 130 (Feb.) 1949.

Seeley reviews the literature on septal perforations and presents a new surgical method of closure. Ten etiologic factors are mentioned. The medical treatment and the already known surgical technic are reviewed. The new procedure is shown in detail. It consists of a double flap repair allowing the continuity of the mucous membrane of the septum to be restored, leaving no raw areas. This is accomplished by mobilizing of the mucous membrane from the cartilaginous and bony portions of the inner nose continuous with that of the septum. The membranous perforation is then sutured and the flap reapplied.

HITSCHLER, Philadelphia.

PROBABLE NASAL DIPHTHERIA, WITH OBSERVATIONS ON SCHICK TESTS. JOSEPH S. FEIBUSH, New York State J. Med. **47**:2600 (Dec. 1) 1947.

Feibush speaks of "the rising incidence of diphtheria noted by observers in Europe, Nova Scotia and upstate New York," and emphasizes "eternal vigilance" in holding this disease in check. The report concerns a 4½ year old boy with a purulent discharge from the left nostril and a thick, yellowish membrane extending from the floor of the nose up along the lateral wall. There was no fever. A culture was reported by the health department to be positive for diphtheria bacilli. Nasal cultures from the patient's father, mother and sister, made at intervals of approximately two weeks, were all negative. Penicillin was given locally by means of drops and nasal packs. A 12,500 unit dose of antitoxin was given intramuscularly. No foreign body was found. There was extension to the throat, a culture was positive for diphtheria bacilli. The author describes Schwarz's reported experiences with the Schick test, which were favorable. His own conclusions are that all children should have the Schick test and a control test three to six months after the last immunizing dose; if the reaction is positive, a course of secondary immunization with alum-precipitated diphtheria toxoid should be given, followed by the Schick test; a combined reaction should result in reim-

munization without a Schick test; the Schick test should be repeated before the child enters school.

[ABSTRACTER'S NOTE.—It is not clear why the author uses the word "probable" in his report. The reported condition seems to be nasal diphtheria, with extension to the throat, as might have been expected. The author delayed for a month examination for a foreign body, which is important in all one-sided purulent nasal conditions, and especially in a case in which there is no fever.]

VOORHEES, New York.

PSYCHOSOMATIC RHINORRHEA AND PSYCHOSOMATIC DYSPNEA. LOUIS STERNBERG, New York State J. Med. **48**:639 (March 15) 1948.

Sternberg, working in the Department of Allergy, Beth Israel Hospital, New York city, differentiates true allergy from the psychosomatic type due to antigen-antibody reactions. Two cases are reported in which there was no antecedent infection although the resemblance to hay fever in 1 and to bronchial asthma in the other was great.

A premedical student with a family history of allergy suffered sneezing and other signs of nasal discomfort during the summer of 1944, at which time she was tense and anxious. The symptoms continued until September, when she became well, to continue so throughout the winter. In the spring of 1945, she entered medical school, began to worry and had a return of all the symptoms. Laboratory tests were negative for pollens and other substances. She soon began to enjoy her studies and did well in examinations; all symptoms disappeared completely during 1946 and 1947.

The second patient had been married at 14 and divorced twice; she had then married a man sixteen years older than herself. Severe asthma developed four years later, becoming worse when the patient quarreled with her husband. Reactions to skin tests were negative. Treatment with autogenous sputum vaccine gave no improvement. The husband died. Eight months later, the patient reported that she had "been perfectly well since." In both cases, the precipitating cause was set down as "psychic stress and strain." Complete recovery occurred when this was relieved.

VOORHEES, New York.

BACTERIAL FILTRATES AND AUTOGENOUS VACCINES IN THE TREATMENT OF CHRONIC SINUS DISEASE AND OTHER CHRONIC RESPIRATORY AFFECTIONS: PRELIMINARY REPORT. HUGH M. KINGHORN, GEORGE E. WILSON and MORRIS DWORSKI, New York State J. Med. **49**:41 (Jan. 1) 1949.

This study was made in Saranac Lake, N. Y. It concerns results obtained with bacterial filtrates and autogenous vaccines in the treatment of "intractable conditions." There were 47 patients suffering from various infections of the respiratory tract—chronic sinusitis and "catarrh"; cough and sputum after surgical treatment of the chest; symptoms such as these where surgical intervention was contraindicated; bronchiectasis, and allergic bronchitis. The laboratory technic of making the agents used is described. It seems complicated and difficult. Injections were intradermal, but an effort was made to plant the agents in the sinuses or in the middle meatus. Staphylococcus was found in all cases! As for the patients who had disease of the lungs, all showed a mixed infection. The tuberculous ones had "far advanced" disease and were "seemingly hopeless . . . nothing else could be done for them!" Of the 47, 1 who had bronchiectasis, did not improve. Two with allergic bronchitis and 6 with far-advanced pulmonary tuberculosis "improved immensely." Of 16 patients who had sinusitis, 81.25 per cent were improved, but 18.75 per cent were unimproved. In discussion, Dr. Howard Westcott, New York,

called for testing of the procedures by others who have similar patients under their care. Skin tests offer no criterion of value of the treatment, since end results only should be decisive. Dr. Edmonde D. Neer, New York, referred to the late Dr. Mackenty's results from surgical treatment of the sinuses, which were excellent. He praised bacteriophage, used locally, and spoke of its "destructive action" on staphylococci. He said, further, that nothing in the literature shows results comparable to the work of Kinghorn and his associates. ". . . only the best laboratory facilities and the most patient investigators can duplicate these results."

VOORHEES, New York.

CONSERVATIVE AND SURGICAL MANAGEMENT OF ETHMOID AND SPHENOID SINUSITIS. STUART L. CRAIG, New York State J. Med. **49**:181 (Jan. 15) 1949.

Conservative treatment calls for (1) dietary considerations, (2) studies as to the supply of vitamins A, B (the complex), C (ascorbic acid) and D, as to environmental conditions and as to worry, fatigue, etc. (3) correction of glandular dyscrasias, hypothyroidism and other endocrinopathies. Glandular preparations are best administered by an endocrinologist. For allergy plus bacterial invasion the physician has such drugs as diphenhydramine hydrochloride (benadryl hydrochloride®) and tripelennamine hydrochloride (pyribenzamine hydrochloride®). Frequent colds in children may be due to obstructing adenoids, tonsils, polypi or deviated septum. Vaccines may be useful against resistant infections of known origin. For diagnosis roentgen study is imperative. Material should be taken from all infected sinuses and cultured. The procedure is outlined. Colon bacilli are often found in the sphenoid sinus! They are not susceptible to penicillin but succumb to streptomycin and sulfadiazine administered singly or in combination. Acute ethmoiditis yields to suction, irrigation, packs, aerosol penicillin, rest in bed, sedation and humidification. Inhalations of menthol, eucalyptus and camphor per formula are valued. Craig also uses a modified Proetz technic. Acute sphenoiditis is often unrecognized. It is suspect whenever pain in areas supplied by the fifth nerve are involved. Irrigations and nasal drops used at home are helpful. Chronic ethmoiditis rarely calls for ablation of the ethmoid labyrinth. Crusts and reinfections are common. The transantral approach does less damage, and the external route may be preferable. Radical operations are permissible in the presence of ocular complications with impending loss of vision. In any case postoperative care is paramount. Use the antibiotics; cauterize excess granulations.

VOORHEES, New York.

Miscellaneous

AN EPIDEMIC OF DIPHTHERIA. WILLIAM A. REILLY, Am. J. Dis. Child. **74**:130 (Aug.) 1947.

Reilly states that there has been a gradual loss of immunity in the United States since 1915, when prophylaxis against diphtheria was begun here. This report deals with 413 personnel of the Fifty-Ninth Evacuation Hospital in Palermo, Sicily, between August 1943 and May 1944. Eighty-one patients had active diphtheria; 116 were carrying virulent diphtheria bacilli but had a mixed infection, and 19 were "healthy carriers." It was difficult to differentiate the infection clinically from follicular tonsillitis or ordinary pharyngitis, for a membrane and toxicity developed in many cases. Patients with negative reactions to the Schick test numbered 13 (16 per cent) of 81 with active diphtheria; this number was higher than previous averages. There were no deaths, but myocarditis developed in 1 man. The frequency of the types of diphtheria organisms was in this order: gravis, mitis, intermedius; however, the mortality was extremely low. Sulfathiazole and sulfadiazine eradicated secondary organisms

and made antitoxin more effective and possibly helped to remove the carrier condition. The duration of the membrane in persons with a positive Schick reaction following diphtheria averaged six and one-half days. The Schick test done within forty-eight hours after administration of 25,000 to 75,000 units of antitoxin gave negative reactions in all cases.

VOORHEES, New York.

IMPORTANCE OF BRONCHIAL RUPTURE IN TUBERCULOSIS OF ENDOTHORACIC LYMPH NODES. O. GÖRGÉNYI-GÖTTCHE and D. KASSAY, Am. J. Dis. Child. **74**: 166 (August) 1947.

The authors, from the St. James and White Cross Hospitals, respectively, in Budapest, Hungary, studied bronchial rupture in 18 children by bronchoscopy and in 8 at autopsy. The percentage of rupture is much higher than hitherto published figures have shown. Ruptures in 52.6 per cent of cases were proved by bronchoscopy; many ruptures occur in the smaller tubes, which cannot be visualized. The prognosis is not so poor as one might think, for the process is one of relief in which debris and bacilli are coughed out. The bronchial rupture takes place four to six months after primary tuberculosis infection becomes manifest; in small babies, it may be earlier.

Prodromal signs are a "ringing brassy" cough, "bitonal" in sound, which is due to vibrating granulation tissue or caseous extrusions, and an "asthmatoid wheeze." Constant fever, with a persistently high sedimentation rate, indicates that the impending rupture is due to the active tuberculous process of the lymph nodes; if fever occurs after the rupture, occlusion of a bronchus or bronchogenic spread is a likely cause. The diagnosis is aided by the Holzknecht-Jakobson sign (movement of the mediastinal organs on inspiration, observed during roentgenologic examination). Fourteen of fifteen ruptures were thus diagnosed. In any case, the sign is an imperative indication for bronchoscopy. Since bronchoscopy is a harmless procedure in infants and children, it is useful, not only in diagnosis, but in the removal of pulmonary masses; it may, therefore, be lifesaving. "Bronchoscopy," say the authors, "is an important procedure which cannot be spared in pediatric practice." The article is forty-one pages in length; it contains two tables and twelve roentgenograms.

VOORHEES, New York.

APHASICS: OBSERVATIONS AND TENTATIVE CONCLUSIONS. JON EISENSON, J. Speech Disorders **12**:290 (Sept.) 1947.

Examinations of persons with aphasia in Army hospitals showed no reliable correlation between the type of language dysfunction and the site of injury. Neither was there any consistent relation between the extent of brain injury and the capacity to relearn. The loss of capacity to abstract observed in civilians with aphasia was by no means a general characteristic of the behavior of most aphasic patients in the Army. An average or higher premorbid intelligence was more frequently associated with aphasia and with the maintenance of aphasic disturbances than was a below average intelligence. Premorbid personality traits are highly important factors. With regard to the site of the brain injury there was only one consistent finding: When aphasic disturbances continued to be present three or four months after the incidence of trauma, it was noticed that the injured area always involved part of the contralateral cerebral hemisphere. Except in cases in which the amount of brain injury was extremely great and involved both cerebral hemispheres, the extent of the injury seemed to have little bearing on the patient's progress and improvement. One patient had suffered approximately a 25 per cent loss of brain tissue in the left occipital and parietal areas. Despite this, he progressed from having almost global aphasia

to a point where he could speak with a fair degree of fluency, could write and copy from dictation and could read simple blueprints. In contrast, another patient, with comparatively little known brain injury in the left temporal area, had persistent and severe difficulties after more than a year of training. In 30 aphasic patients, only 2 showed any significant loss in the ability to abstract. Of 69 aphasic patients at two Army neurosurgical centers, the general classification scores showed that 55 were of average or better intelligence. Data on educational achievement tend to support this observation. A study of 64 patients showed that the majority were of the higher educational level. Subjectively, the author states that aphasic patients who showed severe expressive disturbances had for the most part been withdrawn persons prior to injury. The social service histories of the Red Cross tended to support this observation. The observations, the author states, are tentative.

M. F. PALMER, Wichita, Kan.

SURVEY OF RAGWEED POLLINATION IN THE NEW YORK METROPOLITAN DISTRICT IN 1946. EUGENE H. WALKER, JEROME SHERMAN, ROBERT A. CHAIT and MATTHEW WALZER, New York State J. Med. **47**:1979 (Sept. 15) 1947.

This study was made in the division of allergy, at the Jewish Hospital of Brooklyn, in collaboration with the pollen survey committee of the American Academy of Allergy. The standard technic recommended by the committee was used. In 1946, pollen counts were fairly low for an average of the five boroughs; the count in Manhattan was regarded as most representative. The counts at two stations in Connecticut used as a control were lower than for Greater New York, while those at Washington, D. C., Philadelphia and Cleveland were higher. The peak for the season, at most stations, was on or about September 10. Ragweed pollen in 1946 came in late and was not heavy. In general, weather conditions, such as rain or fog, during the period from May through September affect the scattering of pollen; and, of course, the direction and velocity of winds always have a bearing on the distribution of pollen.

The authors conclude that the available data were insufficient for evaluation of the effect of the campaign carried on by the city authorities for the extermination of ragweed.

VOORHEES, New York.

AN EVALUATION OF ANESTHESIA WITH PENTOTHAL-SODIUM, NITROUS OXIDE, AND ETHER. PAUL W. SEARLES, New York State J. Med. **47**:2091 (Oct. 1) 1947.

Searles's investigations were begun in World War II and have been carried on at in the University of Buffalo since his return. He states that he regards the sequence of thiopental sodium (pentothal sodium®), nitrous oxide and ether as nearly ideal, and states that the barbiturates tend to protect animal organisms against the toxicity of ether. The latter causes a concentration of the blood, and the barbiturates a dilution of the blood. Indications for the combination are operations on the chest and the abdomen, as well as minor procedures. Contraindications are a cardiac history, dyspnea, hepatic lesions, extreme youth or age and alcoholism. Patients with alcoholism may require an overdose, and prolonged postoperative depression may result. Complications are a fall in blood pressure, hiccups, coughing and laryngospasm, all of which can well be controlled by the experienced anesthetist.

The ideal general anesthetic should provide a wide margin of safety, adequate relaxation of the muscles, a quiet operative field, early, pleasant recovery and a minimum of postoperative complications. In Searles's opinion, thiopental sodium secures satisfactory induction and a quiet operative field. Nitrous oxide

plus oxygen sustains the effects mentioned, and ether supplements and prolongs the action of the other anesthetics. The average dose of thiopental sodium is small when used in this combination (from 0.7 to 1.0 Gm.) and much of this is taken up during induction.

VOORHEES, New York.

BRAIN TUMOR SIMULATING PURULENT MENINGITIS. EMANUEL APPELBAUM, JANE W. NORMAN and JOEL J. BRENNER, New York State J. Med. **47**:2106 (Oct. 1) 1947.

A man of 55 was admitted to Bellevue Hospital with severe frontal and nuchal pain of two weeks' duration, stupor and slow cerebration. Nothing abnormal was noted in the examination of the ears, nose and throat. There were bradycardia, slight left facial paresis and positive Brudzinski and Kernig signs. On the fifth day, ophthalmoscopic examination showed engorgement of the vessels and bilateral papilledema. The spinal fluid was full of cells "too numerous to count" (chiefly polymorphonuclear leukocytes), but there were no bacteria. Encephalograms were made, and craniotomy was done on the right temporal lobe. After the operation a slight gain was noted, but the patient soon died. A brain abscess had been suspected, but the actual lesion was a brain tumor. The authors conclude that meningitis sympathica may confuse a diagnosis, particularly when the meningeal condition is pronounced. The condition occurs with brain abscess and with inflammation of the sinuses or the mastoid. The spinal fluid is increased, is hazy to turbid and contains many polymorphonuclear leukocytes and much protein, but the blood sugar is normal, and there are no organisms. Repeated examinations of the spinal fluid may be necessary to assure that the condition is not a delayed bacterial invasion. The authors note the similarities of brain tumor and meningitis as to symptoms. Fever and nuchal rigidity may even appear with cerebral neoplasm, and, rarely, the spinal fluid may become purulent. In this case, the pathologist reported spongioblastoma multiforme, which the authors say may show rapid growth and progessive necrosis. The problem of diagnosis is presented in detail; the prognosis is, of course, uniformly bad.

VOORHEES, New York.

INTRAVENOUS PROCAINE: A PRELIMINARY REPORT. DAVID J. GRAUBARD, RAPHAEL W. ROBERTAZZI and MILTON C. PETERSON, New York State J. Med. **47**:2187 (Oct. 15) 1947.

These studies were undertaken in the anesthesia division of the Reconstruction Hospital Unit, New York Post-Graduate Medical School and Hospital, and reported to the New York state medical society at its meeting in Buffalo, May 7, 1947. Procaine hydrochloride is a toxic drug, and its toxicity is increased when it is used with epinephrine. The authors wished to relieve pain in traumatic and inflammatory conditions. Over 2,000 intravenous infusions of procaine hydrochloride were given with no serious effects. Indications were fractures, traumatic arthritis, the neuritides, congenital spasticity and several other disorders. Reactions were general body warmth, flushing of the head, face and neck, oral dryness, metallic taste, dilatation of the pupils, slight vertigo, general relaxation of the body and alleviation of pain. No permanent results were noted in spinal sclerosis, but there were decreased spasticity, increased coordination and relief of "cold sensations" in poliomyelitis.

Dr. Maurice Bruger, discussing this study, states that sedimentation rates and chemical examinations were made before and during the procaine therapy in 5 cases, but no changes indicating any deleterious effects were observed in the blood or urine. Bruger states: "Procaine administered intravenously twice weekly over

a period of one month in the doses indicated has no measurable effect on renal or hepatic function, . . . sugar or cholesterol content of the blood or the rate of sedimentation." A 0.1 per cent solution in isotonic sodium chloride solution was used. One cubic centimeter contained 1 mg. of procaine hydrochloride.

VOORHEES, New York.

MEDICAL CONSIDERATION OF THE AIR TRAVELER. FREDERICK HOPKINS SHILLITO, New York State J. Med. **47**:2201 (Oct. 15) 1947.

No persisting deleterious effects on the general body economy or on the special sense organs are to be observed under present day conditions of air travel in comparison with earlier days, says Dr. Shillito. Pressurization of the cabin is probably the most important advance; it is now possible to have sea level cabin pressure at 18,000 feet (5,486 M.). The passenger is made comfortable in well upholstered chairs and is served good, hot meals.

As to conditions involving the ears, nose and throat, perforation of the drum is of no concern, but a recently healed perforation may reopen under too abrupt atmospheric change. Infected sinuses may empty on ascent, and the ostiums may close on descent, giving rise to some discomfort. Aerodontalgia (pain in the teeth) does occur. Nasoconstrictor drops are recommended before descent. Either the Politzer or the Valsalva maneuver may be employed to inflate the tubes.

Moderate anemia and cardiac or pulmonary lesions usually are not contraindications for flying. Babies tolerate air travel well. However, there is the possibility of transmitting tropical and other contagious or infectious conditions from one country to another.

VOORHEES, New York.

ESCHERICHIA COLI MENINGITIS TREATED WITH STREPTOMYCIN. HYMAN LIEBERMAN, New York State J. Med. **47**:2206 (Oct. 15) 1947.

This is a rare but usually fatal form of meningitis. In infants under 3 months, this form comprises 30 to 50 per cent of all cases. In older groups, it accounts for 1 case in 300 to 700 (0.3 to 0.14 per cent).

"With the advent of streptomycin," states the author, "the outlook in the treatment of the meningitides due to gram-negative organisms has become considerably brighter." Alexander reported the case of a soldier of 19, wounded in action. Osteomyelitis of the tibia, Escherichia coli bacteremia and Esch. coli meningitis developed. The patient recovered after receiving 18,275,000 units of streptomycin intramuscularly for eleven days, plus 650,000 units intrathecally in thirteen injections. Shields reported recovery in the case of an infant of 5 weeks, whose blood and spinal fluid both contained pure Esch. coli communior. The dosage was 2,630,000 units of streptomycin intramuscularly (30,000 units every three hours for ten days) and 300,000 units intrathecally (30,000 units into the basal cistern daily for ten days). The authors in each case felt that the recovery was due to streptomycin. Lieberman's case was that of a girl of 9 months, who had a normal birth at term, weighing 8 pounds 3 ounces (3,714 Gm.). The diagnosis was made clinically and by culture of the spinal fluid, which showed Esch. coli communior. Large doses of streptomycin were given intramuscularly and intrathecally. Clinical recovery, "somewhat unusual in this type of meningitis," occurred on the sixth day. Because of the falling hemoglobin content, a transfusion of citrated whole blood was given. The total dose of streptomycin was 200,000 micrograms into the spinal canal and 3,400,000 units into the muscles.

VOORHEES, New York.

News and Comment

THIRD ANNUAL CLINICAL SESSION OF THE AMERICAN MEDICAL ASSOCIATION

The Third Annual Clinical Session of the American Medical Association will be held in Washington, D. C., December 6 to 9.

The Clinical Session will provide a full scale scientific program specifically designed for the general practitioner. Outstanding physicians will discuss such subjects as diabetes, pediatrics, laboratory diagnosis, physical medicine and rehabilitation, arthritis, dermatology, roentgen ray diagnosis, cancer and poliomyelitis. Coordinated with this outstanding scientific program will be approximately one hundred scientific exhibits which will present original work on the subjects discussed.

The newest offerings of one hundred and twenty-five manufacturing firms will comprise the Technical Exhibition. Here will be found the latest developments in scientific medical research, drugs and equipment.

Televised surgical and clinical procedures, similar to those shown in color at the Annual Session of the American Medical Association in Atlantic City last June, will be presented at the Washington meeting. The demonstrations will originate in the Johns Hopkins Hospital and will be shown on screens in the Armory. The television schedule will be spread over four days.

The House of Delegates will meet at the Hotel Statler during this session. One of the first orders of business will be the annual selection of the general practitioner who has made an exceptional contribution of service to his community.

An entertainment program is being planned for attending physicians and their wives. The highlight of this program will be on Wednesday evening, December 7, when the Philip Morris Company will originate its "This Is Your Life" broadcast from the Hotel Statler. The radio program will be followed by a stage show, in which outstanding stars will participate.

Blanks for hotel reservations and advance registrations may be found in *The Journal of the American Medical Association.*

SECOND PAN-AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY

The Second Pan-American Congress of Otorhinolaryngology and Bronchoesophagology will take place on Jan. 8 to 15, 1950, in Montevideo and Buenos Aires.

Dr. Justo M. Alonso is president of the congress; Dr. Elías Regules, Dr. Juan C. Munyo and Dr. Juan C. Oreggía are vice presidents, and Dr. Pedro Regules is general secretary. Delegates to the executive committee include Dr. Ricardo Tapia Acuña, of México, D. F., and Dr. Chevalier L. Jackson, of 3401 North Broad Street, Philadelphia. Further information may be obtained from Dr. Pedro Regules by calling San José 1092, Montevideo, Uruguay, or from a delegate of the executive committee.

OTOLARYNGOLOGICAL SOCIETY OF ISRAEL

The Otolaryngological Society of Israel was recently founded, with Dr. Sherman, of Tel-Aviv, as its president. In Israel there are about one hundred ear, nose and throat specialists, from nearly all the countries of the world. The first meeting of the society will be held in Tel-Aviv during the second half of December 1949. The subjects to be discussed at this meeting are the social approach to the problem of deafness and new views on the vestibular apparatus. All colleagues from abroad who wish to participate in the meeting should notify the honorary secretary, Dr. E. Wodak, 9 Javneh Street, Tel-Aviv, Israel, as soon as possible.

Book Reviews

Traitement chirurgical de l'otospongiose. By Maurice Sourdille. Price, 750 francs. Pp. 254, with 52 illustrations. Paris: Masson et Cie, 1948.

This book deals with the work Sourdille has originated and performed over the course of the last twenty years for the surgical treatment of progressive deafness in clinical otosclerosis. He treats this condition with a properly placed labyrinthine fistula which remains sensitive to very slight variations in sound pressures.

Historical data are given on the progressive development of this surgical procedure, on both the experimental and the operative technic. The entire problem of improvement of hearing by labyrinthine operation is presented. It is interesting to note the author's interest in this surgical approach and his early prediction that the surgical steps would be simplified into a one step operation. The historical sequence of the development of surgical relief from deafness is also presented.

The original operative procedures of Bárány, Jenkins, Holmgren, Wittmaack and others are presented and their respective contributions and improvements described.

Sourdille's original presentation on this subject in 1929 is included when he reports his findings and results of his initial surgical endeavors, which were stimulated by his contacts with Holmgren and Bárány in 1924. Also discussed is the surgical development, step by step, by others interested in this work, as well as his own contributions, such as the tympanomeatal plastic flap. The experimental basis and the physioacoustic problems, with the mechanisms involved, such as the role of the head of the malleus, the role of the incus, the functional role of the plastic flap and other factors, are dealt with at length.

Discussion of armamentarium, instrumentation and operative technics is brought up to present day practices and procedures. Postoperative care and complications are clearly discussed.

The author concludes by stating that after his experience of more than twenty years, he firmly believes that the progression of deafness by otosclerosis is definitely retarded or arrested by a properly placed labyrinthine fistula which remains sensitive to very slight variations of pressure. Moreover, the labyrinthine fistula has demonstrated at times a pronounced improvement which is lasting and permanent. The "noli-me-tangere" attitude must now give way to acceptance of this surgical approach, which is withstanding the test of time.

Diseases of the Nose and Throat. Fifth edition. By Sir St. Clair Thomson and V. E. Negus. Price, $16. Pp. 1,024. New York: Appleton-Century-Crofts, Inc., 1949.

The original of this new edition, was described by Chevalier Jackson as "the greatest book on the nose and throat ever published." This edition was started during the adverse circumstances produced in England by the war, and its renowned author, Sir St. Clair Thomson, was about 80 years old when he began the revision. Unfortunately, after working on the book several years, he met with an accident, and much of the work of completing the volume fell to V. E. Negus.

Great maturity of thought and experience is reflected in the material. Major alterations have been made in the revision and recent publications and developments included. Emphasis is placed on physiology, so well investigated by Proetz; less emphasis is placed on the many conditions that are rapidly declining in frequency because of the new methods of treatment. A new chapter on antibiotics, chemotherapy and vitamins is included. Much of the older material is reevaluated in terms of the newer concepts. An excellent chapter is presented on diseases and neoplasms of the nasopharynx, pharynx and larynx. The entire book is organized to present the specific subject and its relation to general principles of medicine and surgery. Many of the narrow limits of specialties are broken down. The relation of systemic diseases, especially blood diseases, to those of the nose and throat is greatly amplified.

The book contains about 1,000 pages and about 60 pages of excellent index. Each chapter is followed by an exceptionally comprehensive bibliography, which is invaluable in investigating the subject. Operative procedures are described in adequate detail. Many of the illustrations are original and give fine orientation to the word descriptions. This new edition is a tribute to the memory of its eminent author and should be used by all rhinolaryngologists.

Treatment of Primary Malignant Tumors of the Maxilla (in French). By Marcel Dargent, Marcel Gignoux and Jean Gaillard. Price, 500 francs. Pp. 216, with 39 illustrations. Paris: Masson & Cie, 1948.

This book is a result of combined work of the tumor center and the department of otolaryngology of the University of Lyons, France. It comprises seven chapters, dealing, respectively, with anatomy, pathology, initial symptomatology, evolution, diagnosis, results of treatment and indications for treatment.

The authors report on 16 cases of sarcoma and 191 cases of epithelioma. The majority of the sarcomas (13) were osteogenic. Of the epitheliomas 44 were of the palate and gum; 43, of anterior localization, with bulging of the cheek; 35 encroached toward the orbit and the ethmoid bone, and 61 showed invasion in all directions. Secondary involvement of the lymph nodes was frequent in the region of the submaxillary gland and the carotid artery (70 cases).

In addition to the regular roentgenographic findings, lateral exposures give useful information about the infiltration of the inferior (palatine) type of tumors.

The authors give preference to total or partial resection of the maxilla, followed by roentgen irradiation or radium treatment and, if indicated, removal of the involved lymph nodes. The treatment with roentgen radiation lasts five to six weeks, with a daily dose of 200 to 300 r in two separate sittings. The radium treatment consists in use of molded apparatus eight hours daily for four to seven days. The authors do not favor electrocoagulation. Forty deaths occurred during the first year; 45 patients were living after two years, and 24 patients were living after five years.

The book is well illustrated with photographs of patients and roentgenograms.

A Contribution to the Study of the Surgical Approach to the Attic By the Endaural Route (in French). By J. Barny de Romanet, M.D. Price not stated. Paris: R. Foulon, 1947.

The author presents the historical development of the postauricular and endaural approaches to the mastoid process. The various incisions for these approaches are described in detail and the advantages and disadvantages of each enumerated.

Romanet believes that the endaural approach is the most advantageous and that there are no real contraindications to it. The main and only real disadvantage is that this approach requires complete surgical competence for complications ensue if it is poorly executed.

In the fenestration operation, if the malleus is to be preserved, as advocated by Sourdille, the postauricular approach is practically imperative.

Whenever the lesion is located in the attic, and in the attic only, the technic of Thies is advocated.

The subject is well presented, and the book is concise and contains a good outline of the various surgical approaches to the mastoid.

Zinc Ions in Ear, Nose and Throat Work. By A. R. Friel. Price, $2. Pp. 59, with illustrations. Bristol, England: John Wright & Sons, Ltd., 1948.

This belated booklet represents the compilation of the experience of a few British enthusiasts on the value of ionization. The first forty pages, over two thirds of the book, give generalities about the use of direct current in medicine, its physiologic effects and the equipment needed for this type of electric therapy.

Considerable stress is laid on the value of the treatment in chronic otorrhea, as practiced by Mr. A. G. Wells, F. R. C. S., in the school clinics of the London County Council. (In 1929 the reviewer observed the extraordinary results that were obtained by Wells and confirmed the work on his return to the United States. Friel gives the technic for treating various kinds of aural infection—with granulations, with different types of perforation, with eczema, etc. The last part of the book is devoted to electrolysis of the turbinate bodies, sinuses and tonsils.

The approach to the subject is direct, thorough and clear. It would have been an advantage if more clinical evidence had been included to support the claims to the value of electrolysis, ionization and coagulation. It would also have been well if more modern and more convenient circuits had been described for the utilization of direct current.

CORRECTION

In the article by Dr. James M. Evans, "Role of Compensatory Hypertrophy and Simple Atrophy in Intranasal Surgery," in the August issue of the ARCHIVES, page 172, the illustration on page 174 should be in the top position of page 176 and the figure in top position on page 176 should be on page 174.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Fourth International Congress of Otolaryngology

President: Dr. V. E. Negus, London, England.
General Secretaries: Dr. F. C. W. Capps and Dr. W. A. Mill, 45 Lincoln's Inn Fields, London, W. C. 2.
Place: London. Time: July 18-23, 1949.

Second Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Prof. Justo Alonso.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Montevideo. Time: January 1950.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. William H. Johnston, 1515 State St., Santa Barbara, Calif.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit 26, Mich.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles 14.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-26, 1950.

American Laryngological Association

President: Dr. Frederick T. Hill, 177 Main St., Waterville, Maine.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Hotel Mark Hopkins, San Francisco. Time: May 23-24, 1950.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Robert L. Moorhead, 125 Remsen St., Brooklyn.
Southern.—Chairman: Dr. William D. Stimson, 899 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. J. Marion Sutherland, 3001 W. Grand Blvd., Detroit.
Western.—Chairman: Dr. J. B. Naftzger, 6777 Hollywood Blvd., Hollywood, Calif.

American Otological Society

President: Dr. Philip E. Meltzer, 20 Charlesgate, W., Boston.
Secretary: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 3, N. Y.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Samuel F. Kelley, 47 E. 61st St., New York 21.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Rea E. Ashley, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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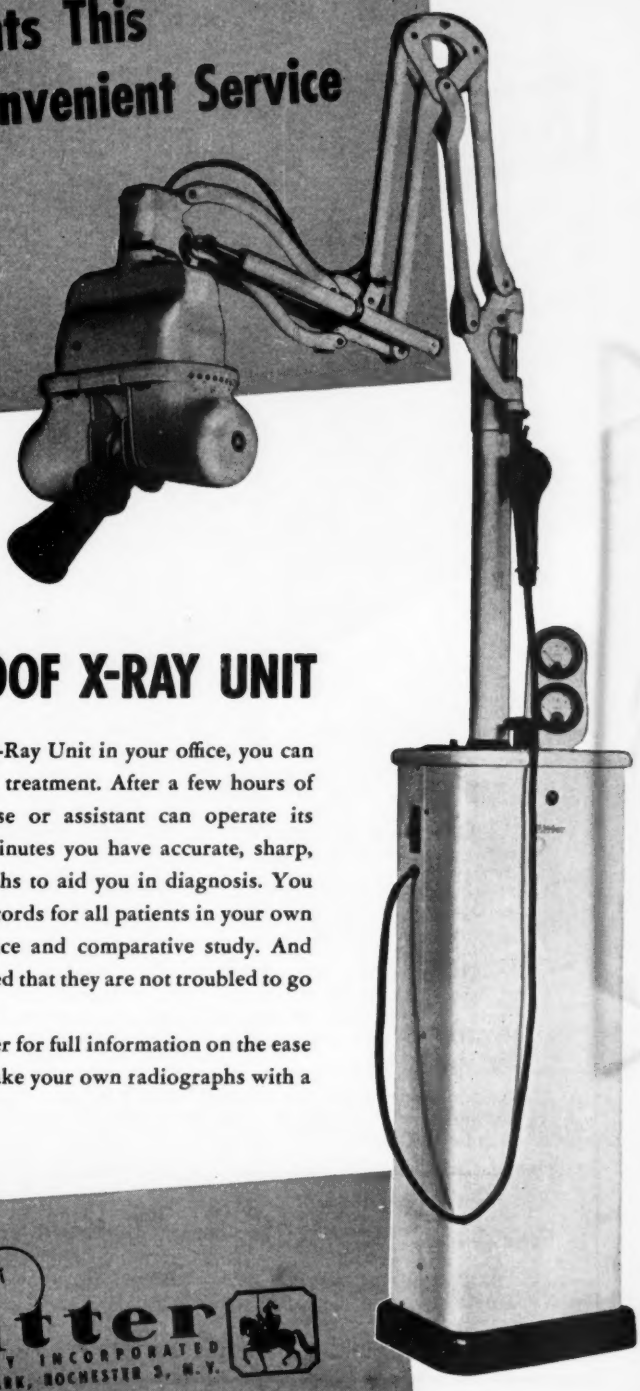

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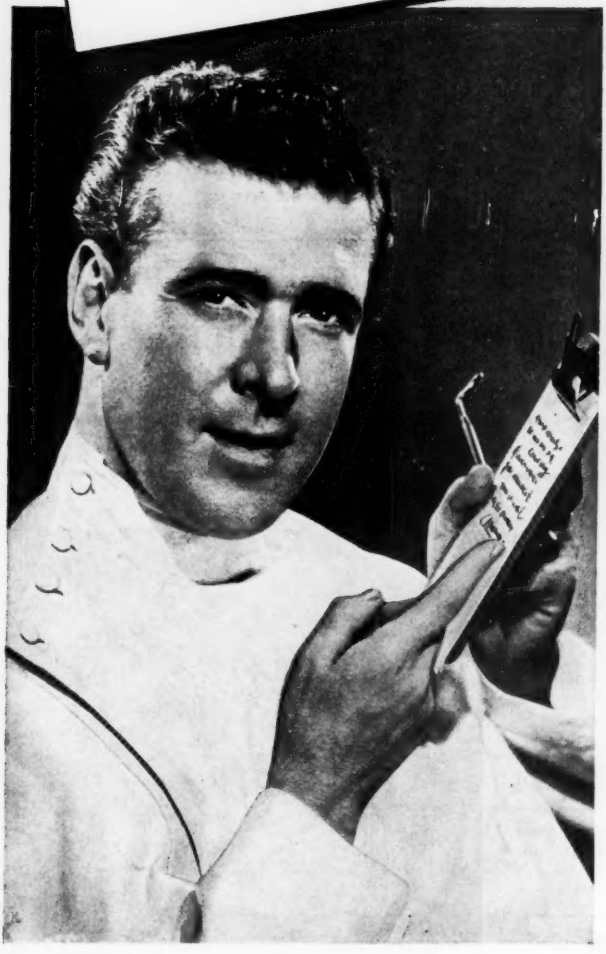

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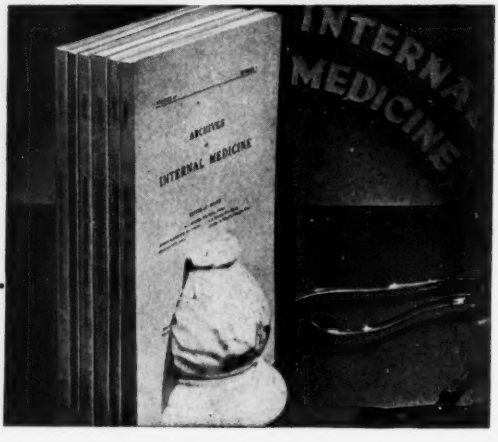

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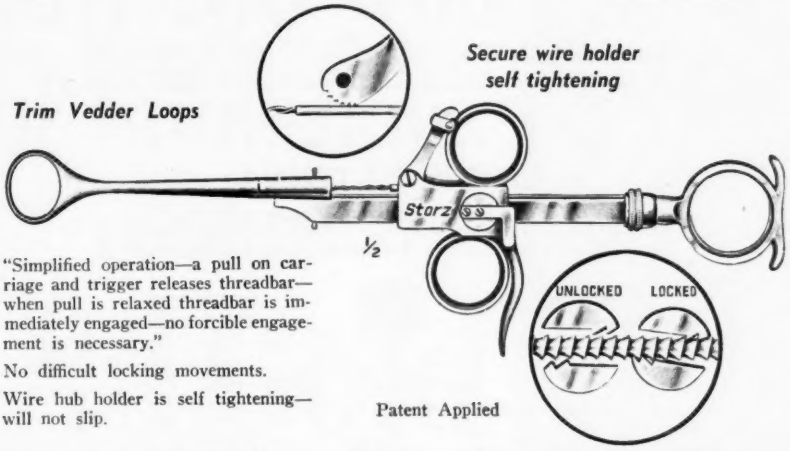

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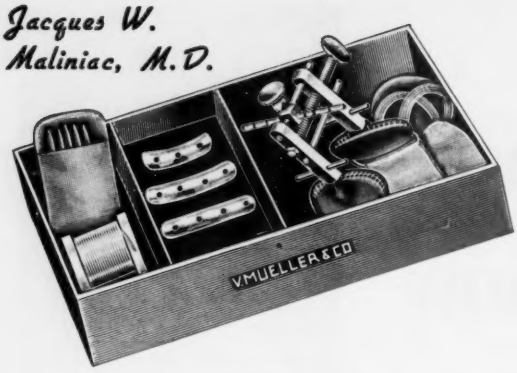

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